A dissertation on

CONDUCTION DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION IN A SERIES OF 250 CASES

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

Chennai.

In partial fulfillment of the regulations
For the award of the degree of

M.D. (GENERAL MEDICINE) BRANCH – I

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI

APRIL 2011

CERTIFICATE

This is to certify that this dissertation entitled

"CONDUCTION DISTURBANCES IN ACUTE
MYOCARDIAL INFARCTION IN A SERIES OF 250 CASES''

Submitted by **Dr. P. Arul,** to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, is in partial fulfillment of the required of the award of M.D. DEGREE BRANCH –I (General Medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of the Unit Chief
(Guide)

Signature of Professor
and HOD.

Signature of Dean

DECLARATION

I, Dr. ARUL.P solemnly declare that the dissertation titled **CONDUCTION DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION IN A SERIES OF 250 CASES**, was done by me at Govt. Stanley medical college and hospital during the year 2010 under the guidance and supervision of my unit chief Prof. P. VIJAYARAGAVAN, M.D.

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of requirement for the award of M.D degree (Branch 1) in general medicine.

Place

Date Dr. ARUL.P.

ACKNOWLEDGEMENT

I owe my thanks to the Dean Govt. Stanley Medical College and Hospital Dr. C. VAMSADHARA, M.D., Ph.D., for allowing me to avail the facilities needed for my dissertation work.

I am grateful to Prof. S. MAGESHKUMAR, M.D., Professor and Head of the department of medicine, Govt. Stanley Medical College and Hospital for permitting me to do the study and for his encouragement.

I express my gratitude to my unit chief Prof. P. VIJAYARAGAVAN, M.D., Prof. G. KARTHIKEYAN, M.D., D.M., and Prof. D. MUTHUKUMAR, M.D., D.M., Department of cardiology, Govt. Stanley Medical College and Hospital for their valuable assistance and guidance.

I am extremely thankful to the registrar Dr. G. VASUMATHI, M.D., and Assistant Professors DR. R. THILAGAVATHY, M.D., DR. S. SUJITH, M.D., for their guidance and encouragement.

I am also thankful to DR. K. TAMILARASAN and DR. S. ILAMARAN, DM (cardiology) post graduates who helped me in conducting this study.

I am particularly thankful to all the ECG technicians for their full co-operation in conducting this study.

Last but not the least, my sincere thanks to all the patients who co-operated for this study, without whom this study could not have been undertaken and to all my colleagues who helped me and shared their knowledge about the study.

CONTENTS

ABBREVATIONS

MI-Myocardial infarction

SA node-Sino atrial node

A-V node-Atrioventricular node

RCA-Right coronary artery

LCA-Left coronary artery

LCX-Left circumflex artery

AV block-Atrioventricular block

RBBB-Right bundle branch block

LBBB-Left bundle branch block

BB-Bifascicular block

IVCD-Intraventricular Conduction Defects

BBB-Bundle Branch Block

LAHB-Left anterior hemiblock

LPFB-Left posterior hemiblock

ALMI-Anterolateral myocardial infarction.

ASMI-Anteroseptal myocardial infarction

EAWMI-Extensive anterior wall myocardial infarction

IWMI-Inferior wall myocardial infarction

IWMI LWMI-Inferior wall with lateral wall myocardial infarction

IWMI PWMI-Inferior wall with posterior wall myocardial infarction

IWMI RVMI-Inferior wall with right ventricular myocardial infarction

INTRODUCTION

Coronary artery heart disease (CAHD) is a disease with multiple causes. It is a disorder with wide and variable spectrum of clinical features.

Ubiquitous in distribution, it is a complex interaction of genetics, environmental factors and life style.

The prevalence of CAHD in India is high and the case burden is rising every years.

Myocardial infarction continues to be a major health problem. About 50% of death from acute myocardial infarction occur within 1 hour of the event and are attributable to arrhythmias. Ischemic injury can produce conduction block at any level of the atrioventricular or intraventricular conduction systems.

The various risk factors causes micro and macrovascular pathology. The possible mechanisms being dyslipidemia, enhanced atherosclerosis, hypertension and diabetic macroangiopathy.

There are reports showing higher incidence of involvement of conduction tissue of heart in diabetes mellitus. Sinus node disorder in diabetes is mostly a manifestation of diabetic autonomic neuropathy.

Hypertension causes an increase in left ventricular mass and fibrous tissue resulting in increased stiffness of the left ventricle leading possibly to reduced coronary reserve, silent myocardial ischemia, and abnormal electrophysiological properties of hypertrophied myocytes and conduction disturbances.

This study is performed to estimate the prevalence of conduction blocks by electrocardiogram in a series of 250 cases of myocardial infarction with respect to various risk factors and outcome.

AIMS OF STUDY

1. To study the incidence of isolated conduction disturbances [without arrhythmias] in acute myocardial infarction in a series of 250 cases admitted in ICCU, Stanley Medical College, Chennai.
2. To study the incidence of conduction disturbances with age and sex distribution and various risk factors.
3. To study the mortality and poor prognosis due to the incidence of conduction disturbances in acute myocardial infarction.

METHODOLOGY

- 250 patients admitted with acute myocardial infarction in intensive coronary care unit from January 2010 were taken up for this study.
- Detailed history was taken and thorough clinical examinations was done as per the proforma given here with enough and available investigations in this hospital to confirm the diagnosis.

INCLUSION CRITERIA

All ST elevation myocardial infarctions.

EXCLUSION CRITERIA

Preexisting conduction blocks and tachyarrhythmias with functional conduction blocks and old myocardial infarctions.

REVIEW OF LITERATURE

ANATOMY AND BLOOD SUPPLY OF THE CONDUCTION SYSTEM

The human heart contains special excitatory and conductive system consisting of

-The SA node

-Inter nodal pathways

-A-V node

-Bundle of HIS

-Right and left bundle branch

-Anterior and posterior fascicle

SA NODE

The SA node is the pace maker of the heart. Is a small flattened ellipsoid strip of specialized muscle about 3mm wide, 15mm long and 1mm thick.

It is situated in the antero superior wall of right atrium at the upper end of sulcus terminalis immediately anterior and lateral to the opening of the superior vena cava. The fibers of SA node are 3 to 5 microns in diameter in contrast to the diameter of 15 to 20 microns of the surrounding atrial muscle fibers.

INTERNODAL PATHWAYS

These are the bundles of atrial fibers that contain the specialized purkinje type fiber conducting the impulses at a higher velocity compared to the surrounding atrial fibers. They connect the SA node and the A-V node. There are three such bundles.

1. Anterior internodal tract of BACHMANN
2. Middle internodal tract of WENCKEBACH
3. Posterior internodal tract of THOREL

ANTERIOR INTERNODAL TRACT

Leaves the anterior margin of SA node passes anterior to the superior vena cava to enter the crest of A-V node.

MIDDLE INTER NODAL TRACT

Leaves the posterior margin of the SA node and passes posterior to the SVC, descends in the interatrial septum and merges with the fibers of anterior internodal tract to enter the crest of A-V node.

POSTERIOR INTERNODAL TRACT

Leaves the posterior margin of the SA node runs with the Crista Terminalis, curves through the valve of IVC to enter the posterior margin of A-V node

ATRIO-VENTRICULAR NODE (A-V NODE)

It is situated in the right atrial side of the interatrial septum just above the opening of the coronary sinus. AV node is continuous with the bundle of HIS.

After leaving the AV node, the bundle of his divides at the juncture of the fibrous and muscular boundaries of the intraventricular septum into the right and left bundle branches. The right bundle branch courses down the right side of intraventricular septum near the endocardium in its upper third, deeper in the muscular portion of the septum in the middle third, and then again near the endocardium in its lower third.

The right bundle does not branch throughout most of its course, but it begins to ramify as it approaches the base of the right anterior papillary muscle with fascicles going to the septal and free wall of the right ventricle.

The main left bundle branch penetrates the membranous portion of the intraventricular septum under the aortic ring. In short course, it divides into two or three fairly discrete branches: (1, 2, 3, 4).

- A predivisional segment.
- An anterior fascicle that crosses the left ventricular outflow tract and terminates in the Purkinje system of the anterolateral wall of the left ventricle.
- A posterior fascicle that fans out extensively inferiorly and posteriorly.
- In about 65 percent of hearts, a fascicle to the intraventricular septum.

Blood Supply –In order to fully understand the relationship between myocardial infarction and dysarrhythmia, it is helpful to review the vascular supply of the different components of the conduction system.

- SA node – supplied by the right coronary artery (RCA) in 60 percent of people: by the left circumflex artery (LCX) in 40 percent.
- AV node – Supplied by the RCA in 90 percent (AV nodal branch); by the LCX in 10 percent of people.
- His bundle – supplied by the RCA (AV nodal branch) with a minor contribution from the septal perforators of the left anterior descending artery (LAD).
- Main or proximal left bundle branch – The LAD coronary artery provides most of the blood supply for the left bundle branch, particularly for the initial portion. There may be some collateral flow from the RCA and LCX systems.
- Left posterior fascicle – The proximal part of the left posterior fascicle is supplied by the AV nodal artery and, at times, by septal branches from the LAD. The distal portion has a dual blood supply from both anterior and posterior septal perforating arteries.
- Left anterior fascicle – The left anterior and mid-septal fascicles are supplied by septal perforators of the LAD and, in about one-half of subjects, by the AV nodal artery.
- Right bundle branch – The right bundle branch receives most of its blood supply from septal perforators from the LAD coronary artery, particularly in its initial course. It also receives some collateral supply from either the RCA of LCX coronary systems, depending upon the dominance of the coronary system.

INNERVATION OF THE HEART

- Is by the cardiac plexus, which is supplied with both sympathetic and parasympathetic fibers. It is situated at the base of the heart and is divided into a superficial and deep part, which are closely connected. Several small ganglia are found in the plexus. The largest and most constant being the cardiac ganglion. The superficial part of the cardiac plexus lies below the arch of the aorta, anterior to the right pulmonary artery.
- The deep part of the cardiac plexus is situated in front of the bifurcation of the trachea. Above the point of division of the pulmonary trunk and posterior to the aortic arch the left coronary plexus gives branches to the left atrium and left ventricle.
- Sympathetic fibers are concerned with cardiac acceleration and dilatation of coronary arteries, while the parasympathetic are concerned with slowing of the heart and constricting the coronary arteries
- The intrinsic cardiac nerve cells are limited to the atria and the inter atrial septum. They are most numerous near the SA node and A-V nodes.

SPREAD OF CARDIAC EXCITATION (5)

Depolarization initiated in SA node spreads radially through the atrium, and then converges on the AV node. Atrial depolarization is complete in 0.1 sec. Because of conduction in AV node is slow, there is a delay of 0.1 sec (AV nodal delay) before excitation spreads to ventricles. From the top of the septum, the wave of depolarization

spreads in the rapidly conducting purkinje fibers to all parts of ventricle in 0.08 to 0.1 sec.

In humans, depolarization of ventricle starts at the left side of the interventricular septum and moves first to the right across the mid portion of septum. The wave of depolarization then spreads down the septum to the apex of the heart. It proceeds from endocardial surface to epicardial surface.

The parts of the heart to be depolarized last are the posterior basal portion of the left ventricle, the pulmonary conus and the upper most portion of the intraventricular septum

ELECTROPHYSIOLOGY OF THE CONDUCTING SYSTEM

Along with nerve, skeletal muscle, and smooth muscle, heart muscle is one of the excitable tissues of the body. It shares any bio electrical properties, with other excitable tissues but as unique electrical properties as well. The electrical activity of the heart is responsible for coordinating the sequence of cardiac activation and contraction. Understanding the normal electrocardiogram requires a substantial knowledge of normal cardiac electrophysiology.

SARCOLEMMA:

The sarcolemma of cardiac cells is comprised of a phospholipid bi layer with its associated membrane glycoproteins. The lipid bi layer membrane is fluid so that the membrane has been likened to a collection of protein icebergs floating in a lipid sea. The

extrinsic proteins often are glycosylated and provide structural support to the sarcolemma. The intrinsic proteins serve as receptors, ion channel and pumps.

IONIC CHANNELS:

Ionic channels are components of the membranes that permit movement of ions across the hydrophobic barrier of the cell membranes. The channels are thought to be membrane spanning proteins that contain water so that hydrated ions can cross the membrane. The channels selective to certain ions, Na+ channel preferentially conducts Na + ions and is less permeable to K+ or Ca2+ ions. Also channels have gating mechanisms which may be open or closed. Depending on the chemicals in the channel or the transmembrane voltage field the degree to which the channel is open may vary. Once open, channels have characteristic open times, one ms for Na + channels and > 100 ms for Ca2+ channels. Each channel seems to operate independently, i.e, the probability that a channel will be open or closed is not influenced by the state of neighbouring channels.

Three different membrane ion channels play an important roles in causing voltage changes of action potential in the cardiac muscle.

-fast sodium channels

-slow calcium - sodium channels

-potassium channels

RESTING TRANSMEMBRANE VOLTAGE:

Cardiac cells have a large transmembrane voltage difference during diastole, about -60 to -90 mV relative to the extracellular fluid potential. The resting transmembrane voltage or potential is an important factor in the electrical behavior of the cell. eg .determining the action potential and also in regulating transmembrane ion transport. In normal cardiac muscle the resting transmembrane concentration gradient for ions such as K+ & Na +are established by active ionic pumping and the membrane conductance for these ions.

THE Na + PUMP:

There is a significant resting Na +influx in cardiac cells. If Na ions are not extruded by the cell, the resting potential would decrease as Na + accumulated. Extrusion of Na+ from the cell requires energy because both the electrical and chemical gradients oppose the removal of Na+. The energy for Na + pumping is provided by the alpha subunit of membrane associated Na-K-ATP ase that extrudes 3 Na + and pumps in 2K+ for each molecule of ATP that is hydrolysed. This enzyme is stimulated by the catecholamines and inhibited by digitalis glycosides. The activity of Na+ pump is electrogenic ie. more positive charge is pumped out than in (a 3:2 Na +-K ratio). Under resting conditions the pump current makes some contribution to the resting membrane potential. Under conditions of increased Na + entry, eg. increased heart rate, pump current will contribute more to diastolic membrane potential driving it to more negative values.

THE CARDIAC ACTION POTENTIAL:

Like other excitable cells the cardiac muscle cells produce an action potential when activated. Of all the excitable cells cardiac cells have the longest action potential, their repolarisation is the slowest. The cardiac purkinje fiber action potential has 5 phases

Phase

0- Rapid depolarization

1- Immediate repolarisation

2- Slow repolarisation /plateau

3- Rapid repolarisation

4- Diastolic interval

Other cardiac cell types have distinctive action potential contours that differ markedly from that of purkinje fibers. They can be thought of as belonging to one or two major groups, fast or slow action potentials.

FAST ACTION POTENTIALS & FAST CONDUCTION:

Most cardiac cells, such as ordinary or specialized atrial fibers, purkinje fibers, and ventricle muscle cells have fast action potentials. These cells have high resting membrane potentials, and when activated, generate a fast rising large amplitude phase 0. This type of phase 0 is associated with very rapid conduction. Cells with fast action potentials tend to have complex and highly developed intercellular connections, the large surface area of complex cellular junctions provide a low resistance pathway for current most highly developed in purkinje fibers and conduction is more rapid in these fibers.

Fast action potentials in the heart are generated by an inward rush of Na+ through an ionic channel that is selectively permeable to Na+ ions when the cells activate. The fast channel activates when membrane potential is rapidly brought from its resting value of -90mV to threshold voltage about -75mV. The Na channel activates quickly, inactivates quickly and has a very high value for maximum ionic conductance when fully activated. The Na + channel is open only for 1-2 ms but the inward Na current intense during that moment. As the resting membrane potential decreases and becomes less and less negative the Na + channel will inactivate more and more so that the inward Na+ current during activation becomes less and less intense. The weaker the Na +current the smaller the amplitude and the rate of rise of phase 0 and the slower impulse conduction will be. At a resting membrane potential of about -60 mV, the Na+ channel is totally inactivated and no response can be elicited even with strong stimuli. This voltage dependent responsiveness of fast action potentials is an important

Factor in arrhythmogenesis and operates in abnormal parts of the heart where cells are depolarized, for instance by local hyperkalemia, by stretch injury or during premature activation. The Na +channel is blocked by antiarrhythmic drugs with class 1 action but not by calcium channel blocking drugs.

SLOW ACTION POTENTIALS & SLOW CONDUCTION:

The action potentials of sinus node P cells & AV nodal cells are very characteristic and quite similar. These cells have a low maximum diastolic value of membrane potential and a small amplitude and relatively slow rising upstroke, ie, phase 0, when

they are activated, potentials are associated with very slow conduction. So there are anatomic reasons for slow conduction in the SA node & AV node. The ionic basis for low potentials is an ionic channel that, when activated, is selectively permeable to calcium (Ca2+) and to a lesser extent to Na+ ions. The channel that carries slow inward current activates at rather positive values of membrane potential such as -40 to – 50 mV. The slow or L type Ca 2+ channel activates slowly, inactivates slowly and has a low value for maximum ionic conductance when fully activated. The conductance of the channel is regulated to some extent by resting membrane potential and Ca2+ and is increased substantially by the catecholamines. The L type Ca 2+ channel can be blocked by drugs such as verapamil, diltiazem or nifedipine. The slow propagation in the sinus and AV nodes permits reentrant excitation to occur in very small areas despite the long refractory period found in cardiac muscle.

SLOW ACTION POTENTIALS IN FAST FIBRES:

Under abnormal circumstances fibers that normally give rise to fast action potentials can develop slow action potentials. Increasing the extracellular K+ concentration to about 16 meq/l will reduce the resting membrane potential of a purkinje fiber to about -50mV and inactivate the Na + channel ;if catecholamine are applied to inactivated purkinje fibers the threshold for calcium channel will shift in the negative direction and the maximum inward Ca 2+ current obtained during activation will increase .under this circumstance electrical stimulation of the purkinje fibers will evoke a slow action potential. Action potentials of acutely ischemic cells resemble the slow responses that occur in high K+, high catecholamine conditions.

Ischemic slow action potentials are abolished by the selective Na + blockers but not Ca 2+ blockers.

REPOLARISATION OF CARDIAC ACTION POTENTIALS:

One of the most striking attributes of the cardiac action potentials is its long plateau, membrane potential remains more positive than -50Mv for several hundred milliseconds. This contrasts with the brief action potentials of peripheral nerves and skeletal muscle fibers which typically last for less than 5 milliseconds. The long plateau provides adequate Ca2+ for contraction and prevents very rapid heart rates. Several ionic mechanisms are known to contribute to the long plateau.

First K+ channels in purkinje fibers show inward going rectification positive to -50mV so that the outward depolarization currents carried by K+ decrease during the plateau of action potential. Second, the cell tends to be held at plateau voltage by inward current carried by Na+ and Ca2+. The slow or secondary inward current normally is triggered by the depolarization caused by phase 0 of the action potential. During the plateau, the small inward and small outward currents are almost perfectly balances so that the membrane potential changes very little for 200 to 400 milliseconds. Phase 3 of the action potential is due to inactivation of I_{ca}-L and activation of I_k, and outward current. As the slow channel inactivates, I_{ca}-L decreases and the cell tends to repolarise.

The K+ accumulation in narrow extracellular cleft may play a role in the depolarization and certainly complicates attempts to study depolarization currents in multi-cellular preparations with voltage clamp techniques.

The normal heart beat is governed by a specialized system that spontaneously generates and distributes each impulse through the heart in a coordinated way. Normally spontaneous impulse generation is much faster in the sinus node than in the His-Purkinje system placing the sinus node in control of the cardiac rhythm.

SPEED OF CONDUCTION

Tissue	**Conduction Rate(m/s)**
SA node	**0.05**
Atrial Pathway	**1**
AV Node	**0.05**
Bundle of HIS	**1**
Purkinje system	**4**
Ventricular Muscle	**1**

ACUTE MYOCARDIAL INFARCTION

Criteria for diagnosis of acute evolving or recent Myocardial Infarction (6)

Either one of the following criteria satisfies the diagnosis of acute, evolving or recent myocardial infarction.

1. Typical rise & gradual fall (troponin) or more rapid rise & fall (CPK MB) biochemical markers of myocardial necrosis with at least one of the following
    a. Ischemic symptoms
    b. Q wave in ECG
    c. ST segment depression or elevation
    d. Coronary artery intervention (e.g., coronary angioplasty)
2. Pathological findings of acute MI

Criteria for established Myocardial Infarctions

Either of the following criteria satisfies the diagnosis for established Myocardial Infarctions.

1. Development of new pathological Q waves on serial ECG readings. The patient may or may not remember previous symptoms.
2. Pathological findings of healed or healing myocardial infarction.

ECG CHANGES

1) Hyper acute phase

The characteristic changes in the leads that are oriented to the infracted surface are as follows (7).

- Increased ventricular activation time.
- Increased amplitude of R wave.
- Slope elevation of ST segment.
- Tall and widened T waves.

2) **Fully evolved phase**

- The myocardial necrosis is represented by QS complex.
- Injury is represented by elevated and coved ST segment.

ELECTROCARDIOGRAPHIC LOCATION OF INFARCTION SITE

Site	***Leads***
➢ Inferior	II, III aV_F
➢ Inferolateral	II, III aV_F,V_4-V_6
➢ True posterior (Posterobasal)	V_1,(reciprocal changes)
➢ Inferoposterior	II, III aV_F,V_1
➢ Anteroseptal	V_1, V_2, V_3
➢ Anterolateral	I, II aV_L,V_4-V_6
➢ Extensive anterior	I,aV_L,V_1-V_6
➢ High Anterolateral	I, aV_L
➢ Anterior (apical)	V_4-V_6,Reciprocal changes inV_1
➢ Posterolateral	V_4R with V_4R-V_6R
➢ Right ventricular	V_1

Atrioventricular (AV) Block

A delay or interruption in conduction of the atrial impulse through specialized AV conducting system.

There are three degrees.

1. First Degree – Delay in conduction.
2. Second Degree – intermittent interruption of conduction
3. Third Degree - Complete interruption of conduction.

FIRST DEGREE AV BLOCK

Delay in conduction reflected by a prolonged PR interval. The PR interval include

i. Time taken for impulse to travel from SA to AV node (usually 0 .03 sec)

ii. Time for the impulse to travel through AV node, bundle of His, bundle branches.

In first degree block, PR interval is prolonged beyond 0.20 sec(0.18 in children)

All the P waves are followed QRS complex.

Associated with

- Coronary artery disease
- Acute rheumatic carditis
- Drugs –Digitalis, beta blockers
- Acute Myocarditis

- Hyperkalemia
- Uremia
- Normal individuals

SECOND DEGREE BLOCK

There is an intermittent failure of AV conduction.

2 TYPES:

1. **Mobitz type I AV block (Wenckebach AV block)**

   The transmission through conducting system becomes increasingly difficult with consecutive beats until it fails and a beat is dropped. P-R interval lengthens with successive beats until a beat fails to be conducted.

   The P-R interval preceding the blocked P is the longest and that follows the blocked P is shortest.

2. **Mobitz type II Atrioventricular Block**

   There is no preceding prolongation of PR interval; PR intervals of all the conducted impulses are normal. It is nearly always due to bilateral bundle branch block; so QRS configuration is abnormal. It frequently progress to complete block.

Causes of second degree block

- Acute rheumatic carditis
- Other acute carditis. eg. Diptheritic
- Coronary artery disease

- Digitalis
- Associated with fast supraventricular rhythms

COMPLETE ATRIOVENTRICULAR BLOCK

All the supraventricular impulses are blocked within the conducting system. The ventricles are activated by a subsidiary ectopic pace maker in the Atrioventricular node below the block or within the ventricles.

Complete block is due to a prolongation of the absolute refractory period so that is occupies the entire cardiac cycle.

Manifestations :

1. ***AV dissociation –***

   P wave bear no relationship to the QRS complexes

2. ***A slow ventricular rate***

   A pace maker situated above the bifurcation of bundle of His and distal to the block will produce a rate of 40 -60/minute.

   If the pace maker is below the common bundle, the rate is about 30 – 40/minute.

3. ***QRS configuration***

   If the pacemaker is above the bifurcation of bundle of His, QRS complex is usually normal in configuration. This is called **AV junctional escape rhythm.**

If the pacemaker is below the bifurcation of common bundle, the QRS configuration is wide and bizarre because the propagation of the impulse in the ventricle occurs in an abnormal fashion. This is termed **ventricular escape rhythm.**

4. *P – P Interval*

When atrial mechanism is sinus, the P – P interval is regular. In 30% cases, the P –P interval which contains the QRS complex is shorter than the P – P interval without QRS complex. This is termed ***ventriculophasic sinus arrhythmia.***

5. R- R interval

This is usually regular

Irregular R- R interval in complete heart block occur in

1. Multiple pacemaker in the AV junction or ventricles
2. Irregular discharge of a single pace maker
3. Premature contractions (Ventricular / AV nodal)
4. Para systole
5. Exit Blocks
6. Intermittent artificial pacemaker induced ventricular rhythm

Causes of complete heart block may be:

1. Acute infections (Diptheria, other bacteria, Viral, Fungal)
2. Electrolyte imbalances
3. Trauma, Cardiac Surgery

4. Drugs – Digitalis
5. Acute inferior wall myocardial infarction.

STOKES – ADAMS ATTACK:

A syncopal attack resulting from ventricular standstill or asystole. This occurs in third degree Av block when the subsidiary ectopic pace maker fails to discharge. This is produced in

1) The transition from second degree to complete heart block.
2) When two or more ectopic pacemakers are in competition.

Syncopal attacks due to paroxysms of ventricular flutter or ventricular fibrillation are also sometimes referred to as Stokes – Adams attack.

RIGHT BUNDLE BRANCH BLOCK

The septum is activated normally from left to right. Left ventricle is activated normally; but right ventricular depolarization is delayed.

COMPLETE RIGHT BUNDLE BRANCH BLOCK

1) Lead V_1 reflects a tall, wide and frequently notched R^1 deflection.
2) The left oriented leads, V_5, V_6 and L_1 reflects a prominent, delayed and widened S wave.
3) QRS duration > 0.12 second.

a) The activation of ventricles begins in the left lower third of interventricular septum and spreads transversely from left to right through septum. This vector is no longer opposed by the normal, smaller right to left septal vector which originate from right bundle branch. So left to right septal vector marginally increase in magnitude. This results in a prominent 'r' in $V_{2.}$
b) Activation of the right side of the interventricular septum and the right and free wall is effected by the activation front which arises in the left side of the septum. This crosses the physiological intraseptal barrier and is conducted through ordinary myocardium.

Consequently it is slow and anomalous and the vector directed to right and anteriorly.

The abnormal right paraseptal vector occurs simultaneously and opposite to the vector of left free wall.

This results in diminution of S wave in V_1; which eventually disappears. There is also attenuation of R wave in left oriented leads.

Abnormal right ventricular activation is reflected by wide R^1 in V_1 and prominent slurred and delayed S in V_6.

So lead V_1 shows a small initial R wave followed by S or s wave of left ventricular depolarization. A terminal bizarre and slurred R^1 wave.

V_5, V_6 and lead I shows

1. A small initial q wave
2. Relatively tall R wave
3. Terminal bizarre and slurred S wave.

INCOMPLETE RIGHT BUNDLE BRANCH BLOCK

Here the conduction through the right bundle branch is delayed but still possible. These manifests as

1. Diminution of the S wave in lead V_2. It is the earliest sign.
2. Slurring of the upstroke of S wave in lead V_2.
3. Appearance of r^1 wave in lead V_2.
4. Amplitude of R^1 wave increases so that configuration is rsR^1 pattern,

QRS deflection is widened, but less than 0.11 sec. and the R^1 deflection is less than 0.04 seconds.

ST segment and T waves in an uncomplicated right bundle branch block shows secondary changes. T wave will be opposite in direction to terminal QRS deflection. ST segment will be slightly convex upwards or minimally depressed.

CAUSES

1. Normal variant
2. Coronary artery disease

3. Acute pulmonary embolism
4. Cardiomyopathies
5. Valvular heart disease
6. Congenital heart disease – Atrial septal defect, Ebstein's anomaly, persistent Atrioventricularis communis.

LEFT BUNDLE BRANCH BLOCK

This is due to the delay or interruption of conduction within the left bundle branch.

There are three components of ventricular activation in complete left bundle branch block:

1. Right septal activation.

The normal small right septal vector is not opposed by a concomitant, greater, left septal vector. This results in:

a. A small initial positive deflection in left oriented leads.

b. A small negative deflection in right oriented leads.

2. Delayed and anomalous left septal activation.

The right septal activation process jump an intraseptal physiological barrier and activates the left side of septum in an anomalous manner,The vector is directed posteriorly and to the left.

This produces a tall R^1 in left oriented leads.

3. Delayed and anomalous activation of free left ventricular wall.

This results in a vector directed posteriorly, left and somewhat superiorly. This produces a tall R^1 deflection in left oriented leads and a deep S wave in right oriented leads.

ELECTROCARDIOGRAPHIC MANIFESTATIONS

a. QRS duration more than 0.12 sec.
b. QRS in various leads

V5, V6 and lead 1 usually presents a tall and notched R wave-an RR^1 or M shaped complex. The intrinsicoid deflection may be 0.09 to 0.10 seconds.

Lead V1, V2 presents a widened, notched QS complex or an Rs complex.

ST segment and T wave shows secondary changes; in the opposite direction of terminal segment.

INCOMPLETE LEFT BUNDLE BRANCH BLOCK

1. The small initial q wave in lead V5, V6 and lead1 disappears, resulting in a single tall R wave.

2. Small initial r wave in lead v1 disappears. On further progression, a slur appears on the upstroke of QRS complex, accompanied by widening and then notching of QRS .QRS duration will be less than 0.12 second.

CAUSES

1. Coronary artery disease

2. Hypertension

3. Aortic valve disease

4. Cardiomyopathy

LEFT ANTERIOR FASICULAR BLOCK

CRITERIA FOR DIAGNOSIS:

1. Abnormal left axis deviation (usually between -45° and -60°)

2 .rS complexes in lead II, III, aVF and qr complexes in lead I and aV L

3. Delayed intrisicoid deflection in lead I and aVL(>0.045second)

4. Peak of r wave in lead III occurring earlier than peak of r wave in lead II.

5. Peak of R wave in lead aVL occurring earlier than peak of Rwave in aVR

6. Increased QRS voltage in limb leads.

CAUSES

Normal variant

Coronary artery heart disease

Left ventricular hypertrophy

LEFT POSTERIOR FASCICULAR BLOCK

CRITERIA FOR DIAGNOSIS:

1. Right axis deviation

2. r in lead I and aVL ,q in II ,III and aVF

3. Delayed intrinsicoid deflection in aVF (>.045 second)

4. Increased QRS voltage in limb leads.

5. No evidence of right ventricular hypertrophy.

A smaller sequence of ventricular activation can also occur in right ventricular hypertrophy, pleuropulmonary disease and extremely vertical anatomical heart position. So a diagnosis of pure posterior fascicular block cannot be made from ECG alone.

RISK FACTORS:

Non modifiable risk factors are

1. Age
2. Male sex
3. Family history of premature coronary artery disease
4. Angiotensin converting enzyme polymorphism

Modifiable risk factors

1. Hypertension
2. Diabetes
3. Low HDL level
4. Cigarette smoking
5. Physical inactivity
6. Obesity

7. Hyperlipidemia
8. Hyperfibrinogenemia
9. Hyper homocystenemia

a) Age and Sex

Obviously these risk factors cannot be corrected; however there is considerable evidence that hormone replacement therapy may reduce the risk of heart disease in post menopausal women.

b) Family history

Coronary Artery Disease (CAD) runs in families. This may be due to genetic factors or the effect of shared environment. At present it is estimated that about 40% of risk is controlled by genetic factors & 60% by environmental factors.

c) Hypertension

Several major prospective epidemiological studies have found that both systolic and diastolic hypertension have strong relationship to CHD (8). A metaanalysis of 17 randomized trials of antihypertensive drugs to patients with mild to moderate Hypertension has found that the risk of CHD was lowered by 10%. Blood pressure can be lowered by weight loss, exercise, salt restriction, avoidance of alcohol and drugs.

Left Ventricular Hypertrophy:

It is defined by either electrocardiogram or echocardiography. It is an important independent risk factor roughly doubling the risk for cardiovascular death in both men and women (9). Left ventricular hypertrophy is associated with obesity, excessive salt intake, advanced age heredity (10). Several studies have found that angiotensin converting enzyme inhibitors reduces left ventricular mass by 12%, Calcium channel blockers by 11%, beta blockers by 5% and diuretics by 8%.

d) Obesity

Obesity is defined as Body Mass index >= 30, is a major risk factor for CAD (11) it is associated with insulin resistance, hyperinsulinemia, Type I DM, systemic hypertension, low HDL, hypertriglyceridemia and left ventricular hypertrophy (12).

e) Homocystinuria

Hyperhomocystinuria is an independent risk factor for CAD. A series of studies have found linear dose response relationship between plasma homocysteine levels and a mortality rate of 4.5% for patients with higher levels of homocysteine. Nutritional supplementation with folic acid can lower homocysteine levels in many individuals.

f) Alcohol

Mild to moderate alcohol consumption (2-4 units/day) is associated with reduced rates of CHD. However heavy drinking is associated with hypertension and excess cardiac events.

g) Infections/ Inflammation

Recent publications have furnished evidence in support of a role for Chlamydia pneumonia, CMV, or other infections agents in CAD.

h) Metabolic Syndrome

Metabolic Syndrome a conditions characterized by metabolic risk factors and increased risk of coronary heart disease. The metabolic syndrome can be said to present, when a person has three of the following;

1. Abdominal obesity (waist circumference > 102 cm in males, >88 cm in females)
2. Triglycerides > 150mg/dl
3. HDL>40mg/dl in males and >50 mg/dl in females
4. BP > 130/85 mm HG
5. Fasting glucose >110mg /dl

i) Physical inactivity

It is an independent risk factor for CHD roughly doubles the risk (13). There is a dose response relationship between the amount of exercise performed

weekly and death from cardiovascular disease and all causes. Physical activity slows the progression of atherosclerosis in human.

j) Psychological factors

The role of personality, environment, social support, social contact, stress, depression has all been associated with increased risk for Coronary Heart Disease.

The absolute risk for development of CAD over the next decade can be estimated for men & women by FRAMINGHAM RISK tables (14).

(K) Smoking

A strong dose response relationship between cigarette smoking and CHD has been observed in both sexes, in the young, in the elderly and in all racial groups (15). There is no evidence that filters or other modification of cigarettes reduces the risk (16).

Exposure to environmental tobacco smoke or passive smoking has been recognized is a modifiable risk factor (17). Exposure to tobacco smoke by non smokes was consistently associated with a 20 to 30% increase in risk. The increased risk in smokers is due to their increased vascular reactivity. Smokers have increased levels of fibrinogen and increased platelet aggregrability.

CARDIAC INVOLVEMENT IN DIABETES MELLITUS

Heart has long been known to be involved in a number of ways by diabetes mellitus. These are

1. Ischaemic heart disease.
2. Hypertensive heart disease.
3. Diabetic cardiomyopathy
4. Conducting tissue involvement

   The diabetic population suffers the following draw backs

   a. Increased risk of infarction by 2 to 3 times

   b. The protective effect of being premenopausal women is lost

   c. Silent myocardial infarction

   d. Higher morbidity

   e. Less prominent circadian rhythm of attack

   f. Excess cardiovascular and all cause mortality

Conduction disturbances are well recognized complications of acute Myocardial infarction. They are induced by either autonomic imbalance or ischemia and necrosis of the conduction system. It is important to recognize which situations are transient and which are likely to progress to irreversible and symptomatic heart block.

The frequency of high degree block with acute Myocardial infarction. The available data concerning the frequency of heart block after acute frequency of heart block after acute MI are largely derived from studies performed before the acute revascularization strategy of the 1990 s (18, 19, 20, 21, 22).

Reports from later thrombolytic trials suggest that the incidence has not changed. The general incidence of Intraventricular conduction disturbances during an acute MI is 10 -12% (23, 24, 25).

New Bifascicular block was associated with 31% risk of CHB.

The development of CHB was associated with a poor prognosis. Affected patients had a 28% rise of sudden cardiac death or recurrent high degree AV block during the first year of follow up.

Scoring system to predict the development of CHB in patients with acute MI (26). The risk of developing CHB in this report was not associated with infarct location or left ventricular ejection fraction. One point was assigned for the new development of PR prolongation second degree AV stock, LAFB or LPFB, LBBB and RBBB. The risk of progression to CHB was:

1.2% to 6.8% with a point score of 0,

7.8 to 10% with a score of 1.

25% to 30% with a score of 2.

36% with a score of 3 or more.

This scoring system was then applied to six previously published studies of acute MI complicated by CHB; a similar correlation was seen.

MORTALITY:

Although high degree AV block in patients with an inferior MI is usually transient, it is associated with increased in-hospital mortality. In the prethrombolytic era, the average mortality was 9 percent without AV block compared to 23 and 29 percent when second and third degree AV block were present, respectively (27,28, 29) among patients treated with a thrombolytic agent, the in-hospital mortality with AV block is 7 to 20 percent versus 2 to 4 percent in the absence of high-grade AV block.

The occurrence of high degree Atrioventricular block in patients with an anterior wall myocardial infarction is associated with a higher increase in in-hospital mortality than is seen with an inferior wall infarction. This is because of more extensive myocardial involvement and a higher incidence of hemodynamic complications when high degree AV block is associated with an anterior wall infarction.

PACEMAKER THERAPY

The point system described above can be useful in deciding which patients might benefit most from cardiac pacing (26). Patients with two or more of the following new

findings are at 25 to 36 percent risk of progression to fascicular block, LBBB, and RBBB.

We use the classifications for temporary pacemakers as recommended by the AHA/ACC Committee on Pacemaker Implantation and the recent AHA/ACC guidelines for management of acute myocardial infarction. One concern is that 10 to 20 percent of patients develop complications from the insertion of pacemakers including infection, perforation, and arrhythmias.

Indications for temporary transvenous pacing – Temporary transvenous pacing is suggested for patients at risk of developing CHB as a consequence of acute MI. We do not favor the use of the external temporary cardiac pacer in acute MI because of the physical discomfort associated with external pacing. The following are the AHA/ACC recommendations concerning temporary cardiac pacing for conduction disturbances in acute MI followed by comments related to our clinical approach (30).

CLASS I

Conditions for which there is general agreement that temporary pacemakers should be implanted.

A. Complete (third – degree) heart block.
B. Bilateral BBB that includes alternating right and left BBB or RBBB with alternating LAFB and LPFB of any age.
C. New or age indeterminate bifascicular block (RBBB with LAFB or LPFB or LBBB) with first – degree AV block.
D. Asystole.

E. Symptomatic bradycardia that includes sinus bradycadia with hypotension and type I second degree AV block with hypertension not responsive to atropine.
F. Mobitz type II second-degree AV block.

CLASS II

Conditions for which temporary pacemakers are frequently used but there is divergence of opinion with respect to the necessity of their insertion. Class IIa are conditions for which the weight of evidence or opinion is in favor of usefulness of efficacy. Class IIb are conditions for which the usefulness or efficacy are less well established.

A. Mobitz type I with bradycardia or with hemodynamic consequence and hypotension.
B. Preexisting bifascicular block.
C. Preexisting bifascicular block with first degree AV block.
D. New LBBB.

Class IIa

A. New or age indeterminate RBBB with LAFB or LPFB

B. RBBB with first-degree AV block.

C. New or age indeterminate LBBB

D. Recurrent sinus pauses (greater than three seconds) not responsive to atropine

E. Incessant ventricular tachycardia for atrial or ventricular overdrive pacing

Class IIb

A. Bifascicular block of indeterminate age

B.New or age indeterminate isolated RBBB

Class III – Conditions for which there is general agreement that temporary pacemakers are unnecessary.

A. First-degree heart block

B. Mobitz type I second-degree AV block with normal hemodynamics

C. Accelerated idioventricular rhythm

D. BBB or fascicular block know to exist before the acute MI

Our approach varies with the location of the infarct. We implant transvenous pacemakers in inferior infarcts for:

- Symptoms or worsening ischemia related to bradycardia and hypotension.
- Bradycardia-induced tachyarrhythmias

We do not, for example, insert a pacemaker for complete heart block with narrow QRS and an adequate ventricular rate in this setting, since the junctional rhythm is stable.

In anterior infarcts, we insert a transvenous pacemaker for all of the Class I indications in the AHA/ACC guidelines plus symptomatic bradyyarrhythmias of any type, including those that cause tachyarrhythmias. We do not pace for the Class IIb indications and some of the Class IIa indications, particularly A and B. The optimal approach to C, a new or age indetermine LBBB, is uncertain: it is recommended that a pacemaker be inserted in this setting by one of the authors (Dr Arnsdorf) but not by the other two (Drs Zimetbaum and Josephson).

Indications for permanent pacing – we follow the recommendations of AHA/ACC committee on Pacemaker Implantation regarding the implantation of permanent pacemakers after MI (31).

Class I – conditions for which there is general agreement that permanent pacemakers should be implanted.

A. Persistent advanced second degree (Mobitz type 2) AV block or complete heart block with block in the His-Purkinje system (bilateral bundle branch block). We interpret this recommendation in terms of the trifascicular conduction system and include persistent RBBB with LAFB, RBBB with LPFB, alternating right and left bundle branch block, and LBBB with PR prolongation.
B. Patients with transient advanced AV block (second-or third-degree) and associated bundle branch block
C. Patients with symptomatic AV block at any level

Class II – Conditions for which permanent pacemakers are frequently used but there is divergence of opinion with respect to the necessity of their insertion.

Patients with persistent advanced block at the AV node. This is generally a Wenckebach type of conduction disorder, which is important only if it results in a slow rate and/or hemodynamic impairment. If such an abnormality persists, however, one must suspect disease in both the right and left coronary systems, and further investigation may be interrupted.

Class III – Conditions for which there is general agreement that pacemakers are unnecessary.

A. Transient AV conduction disturbances in the absence of intraventricular conduction defects.
B. Transient AV block in the presence of isolated LAFB.
C. Acquired LAFB in the absence of AV block.
D. Persistent first degree AV block in the presence of BBB not demonstrated previously.

OBSERVATION AND RESULTS

250 Patients of acute myocardial infarction were included in this study. They consisted of 167 male and 83 female patients. The youngest patient was a 23 year old male and the oldest patient was an 84 year old female.

1. INCIDENCE OF HEART BLOCKS IN PATIENTS WITH ACUTE MI.

No of patients with heart blocks	No of patients without heart blocks	Total patients
51	199	250

Out of 250 patients with acute MI, 51 patients had heart blocks.

2. INCIDENCE OF HEART BLOCKS IN PATIENTS WITH ACUTE MI IN ACCORDANCE TO AGE

Age	No.	No of pts with Heart block	%	P-Value
<=30	3	0	0.0%	Chi-Square 0.881 Not Sig
31-40	33	8	24.2%	
41-50	75	16	21.3%	
51-60	65	11	16.9%	
61-70	58	13	22.4%	
>70	16	3	18.8%	

The maximum incidence of MI is in the age group of 41 to 50 years. In this study the heart blocks are more common in the age group of 31 to 40 years.

3. INCIDENCE OF HEART BLOCKS IN PATIENTS WITH ACUTE MI IN ACCORDANCE WITH SEX

Gender	No.	No of pt with Heart block	%	P-Value
Male	167	33	19.8%	Chi-Square 0.722 Not Sig
Female	83	18	21.7%	

Out of 250 patients 167 were male and 83 females. Out of these 33 males and 18 females had heart blocks.

4. INCIDENCE OF HEART BLOCKS I N PATIENTS WITH VARIOUS RISK FACTORS IN ACUTE MI

Risk factors	No of pts	No of pt with Heart block	%	P-Value
Smoking	109	22	20.2%	0.94
Alcohol	85	20	23.5%	0.378
Diabetes	74	21	28.4%	0.042
Hypertension	61	11	18.0%	0.598
High Cholesterol	79	18	22.8%	0.525

It is found that among various risk factors diabetic patients had the highest percentage of heart blocks followed by alcoholics and patients with high cholesterol.

5. THE INCIDENCE OF TYPES OF MI.

TYPE OF MI	No.	%
ALMI	25	10.0%
ASMI	109	43.6%
EAWMI	13	5.2%
IWMI	44	17.6%
IWMI LWMI	2	0.8%
IWMI PWMI	36	14.4%
IWMI RVMI	21	8.4%

The incidence of AWMI is more than IWMI. The incidence of AWMI was 58.8%and that of IWMI was 41.2%

6. INCIDENCE OF DIFFERENT TYPES OF HEART BLOCKS IN ACUTE MI

AV BLOCKS 12

AVBLOCK	No.	%
1 DEG	5	41.7%
2 DEG	1	8.3%
3 DEG	6	50.0%

IVCD 39

IVCD	No.	%
CHB	2	5.1%
LAHB	7	17.9%
LBBB	13	33.3%
RBBB	17	43.6%

RBBB is the most commonly occurring Intraventricular conduction defect (43.6%)

7. INCIDENCE OF BLOCKS IN RELATION TO SITE OF MI.

TYPE OF MI	AVBLOCK			IVCD				Total
	1 DEG	2 DEG	3 DEG	BB	LAHB	LBBB	RBBB	
ALMI	0	0	0	2	1	2	2	7
ASMI	0	0	1	0	5	8	7	21
EAWMI	0	0	0	0	0	0	1	1
IWMI	0	1	1	0	0	3	3	8
IWMI PWMI	3	0	2	0	0	0	2	7
IWMI RVMI	2	0	2	0	1	0	2	7
Total	5	1	6	2	7	13	17	51

The number of AV blocks is 5 in AWMI and 7 in IWMI

Out of 250 patients with acute MI 39 had intraventricular conduction defect, out of which 25 occurred in patients with AWMI and 14 in patients with IWMI.

8. OUTCOME

TOTAL NO OF PATIENTS	NO OF PATIENTS EXPIRED	%
250	26	10.4

The total mortality is 10.4% of all patients with acute MI.

9. OUTCOME WITH REFERENCE TO TYPE OF MI

TYPE OF MI	No.	PERCENTAGE	DEATH	PERCENTAGE
ALMI	25	10%	4	15.3%
ASMI	109	43%	12	46.1%
EAWMI	13	5.2%	3	11.5%
IWMI	43	17.2%	1	3.8%
IWMI LWMI	2	0.8%	0	0%
IWMI PWMI	35	14%	2	7.6%
IWMI RVMI	23	9.2%	4	15.3%

The number of deaths is high in AWMI when compared to IWMI.

10. OUTCOME WITH REFERENCE TO HEART BLOCKS

TYPE OF BLOCK	NO	DEATH	PERCENTAGE
1 DEG	5	0	0%
2 DEG	1	0	0%
3 DEG	6	3	50%
RBBB	17	8	47%
LBBB	13	4	30.7%
LAFB	7	2	28.5%
BB	2	1	50%

The mortality among patients with heart block is 69.2% and patients without heart blocks is 30.8%

The mortality among patients with third degree heart block and bifascicular block is 50% and that of RBBB is 47%.

11. DISTRIBUTION OF DEATHS IN AV BLOCKS

Type of block	No. of patients	Death	Percentage
1 DEG	5	0	0%
2 DEG	1	0	0%
3 DEG	6	3	50%

The mortality is highest in third degree AV blocks..

DISTRIBUTION OF DEATHS IN IVCD

IVCD	NO OF PATIENTS	NO OF DEATHS	%
RBBB	17	1	5.8
LBBB	13	4	30.7
LAFB	7	2	28.5
BB	2	1	50

The mortality rate is 50% in bifasicular blocks, 30.7% in left bundle branch blocks, and 28.5% in left anterior fasicular blocks.

DISCUSSION

1. AGE AND SEX.

In this study that included 250 patients of acute myocardial infarction, the maximum incidence of acute MI occurred in the age group of 41 to 50 .The youngest patient was a 23 year old male and oldest was 84 year old female. The incidence of serious arrthymias remain constant at various ages (32)

In this study male patients constituted 66.8% of study group. Female population constituted 33.2% of study group. According to literature males in the age group of 40 to 70 are prone for MI (33), which correlates with our study.

2. INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH RISK FACTORS

Smoking was present in 109 among 250 patients with acute MI. It is the most common risk factor in our study group. Next common risk factor is alcohol.

In our study out of 250 patients with acute MI, 61 had high blood pressure and 79 had high cholesterol (> 200 mgm/dl). According to LAW et al a 10% increase in cholesterol is associated with 20- 30 % increase in risk of coronary artery disease.

In our study, the association between diabetes and heart blocks was 28.4% and P value is 0.042, found to be significant. In 1976, Dutta et al (34) did a study on 817 diabetics without myocardial infarction. 50 patients had bundle branch blocks.

A study by Rogdrigvez- Morci. M.et al (35) showed that of the 1990 type 2 diabetics 29.1% showed arrhythmias in ECG

Thraindottir I.S. (36) et al in a long prospective study involving 9135 males & 9627 females showed that there was significant relation between right bundle branch block and diabetes.

Panja and Dutta (37) et al in a study of 300 cases of complete heart block showed that diabetes was present in 14.6%.

Partiman and Bradley(38) were among the first to describe high incidence of bundle branch block in the diabetic myocardial infarction.

The Framingham study has shown that the incidence of arrhythmias, conduction disturbances and sudden death is considerably higher among hypertensives. The sub endocardial fibrosis may be the involvement of conduction pathway.

A review of hypertension and LVH by Franz H. Messerlt et al, in the cardiology clinics (NOV 1995) states that the increased incidence of conduction disturbances in hypertensive is probably due to increased fibrous tissue or altered collagen content.

Gopinath et al in 1994 in his study on 307 patients with hypertension found 11% had conduction disturbances. Our study correlates well with the above study

3. INCIDENCE OF RHYTHM DISTURBANCES IN RELATION TO LOCATION OF ACUTE MYOCARDIAL INFARCTION

According to literature overall incidence of conduction blocks in acute MI is 12-25 % (39).The results of our study coincides with literature. In our study the incidence of heart blocks is 20. 4% .Also literature say AV blocks are more common in IWMI than AWMI (40). If it occurs in AWMI it is associated with extensive myocardial necrosis and poor prognosis.

In our study there were 7 patients developing AV block in AWMI. This also correlates with the literature. According to Braunwald's text book of cardiology the incidence of MOBITZ type two block is less than 1%. In our study there was no patient with MOBITZ type two block . In the study of Atkins and his group the most common abnormal conduction pattern was RBBB followed by LAFB and LBBB.In our study
The incidence of RBBB ,LAFB&LBBB are 17,7 and 13 respectively.Thus RBBB is the most common abnormal conduction block.

A study in 432 consecutive patients with acute MI and BBB showed BBB was 3 times as likely to be associated with AWMI, than IWMI.
Another study among 681 patients from one institution entered in to the TAMI-9 or GUSTO 1 trial.

1. It showed incidence of transient BBB was 18.4%, while persistent BBB occurred in 5.3% of patients

2. RBBB was most common 13%, followed by LBBB (7%)

3. Mortality was higher in patients with BBB

In our study two patients developed bifasicular block .No patient developed LPFB.

Thus the anatomical characteristic of Right bundle branch and anterior fascicle of Left bundle branch, their length and slenderness make them more vulnerable to ischemic injury than the more compact posterior fascicle of the left bundle branch. Their initial common course and blood supply account for the high incidence of RBBB and LAFB in the setting of acute MI.

The mortality in patients with heart block is more than the mortality in patients without heart block (41). In our study it is 69.2 % and 30.8 % respectively.

CONCLUSION

1. The incidence of heart blocks in acute MI is 20.4 %

2. The distribution of heart blocks is almost equal in both sexes.

3. Heart blocks are more common in the age group of 31 to 40 years.

4. The incidence of heart blocks in diabetes mellitus and alcohol are high (28.4% and 23.5% respectively)

5. AV blocks are more common in IWMI

6. Most of the IVCD occur in patients with AWMI

7. RBBB is the most common IVCD with incidence of 43.6%

8. Mortality is high in patients with third degree AV block and bifasicular block.

BIBLIOGRAPHY

1. Gray's text book of anatomy 19th edition pp138
2. Rosenbaun M.elizari , MV Lazzari, Jo . The Hemiblocks.Tampa Tracings . Tamba 1970
3. Hecht , HH, Kossman, CE, Atrioventricular and intraventricular conduction; Revised nomenclature and concepts: AMJ Cardio-1973;31-232
4. Demoolin , JC,Kulbertus HE Histopathological examination of the concept of left Hemiblock .Br.Heart Journal 1972; 34; 807
5. William F. Ganong Review of medical physiology 21 st edition.Chapter 28 pp 551-552
6. Alpert et al Myocardial infarction redefined ; a concensus document of the joint Europian society of cardiology for the redefleition of myocardial infarction 2000
7. Lee Shamroth an introduction to electrocardiography: 7th edition 1990:pp137
8. Macmohan et al bood pressure stroke and coronary artery heart disease ; part 1 prolonged differences in blood pressure Lancet 1990. 335 pp765
9. Levy D et al prognostic implication of echocardiography determined left ventricular mass in the Framingham heart study N Eyl J Med 1990 ; 322;pp156
10. Hariji KJ potential new cardiovascular risk factors; left ventricular hypertrophy ; AM Intern medicine 1999:131 pp376
11. Eckel et al obesity as a major risk factor, circulation 1998, 97:pp2099
12. Molodad et al prevalence of obesity related risk factors 2001, JAMA 2003: 289 pp76-79

13. Fletcher GDF et al statements on exercise benefits and recommendations for physical activity .circulation 1996,94:pp857
14. Thirt report of the national cholesterol educations programs (NCEP) expert panel on detection evaluation and treatment on high cholesterol in adults circulation 2002 106 pp 3143-3421
15. US department of health and human services the health consequences of smoking; cardiovascular disease. A report of the surgeon general Washington DC 1983
16. Castelli et al, the filter cigarette and coronary heart disease. The Framington story lancet 1981 2; pp109
17. Fielding JE phenow KJ health effects of smoking, N Eng J Med 1988. 319 pp1452
18. Godman, MJ Lasser BW Julian, DJ, complete bundle branch block complicating acute myocardial infarction. N Eng J Medicine 1970; 282;287
19. Gupta P.Heart block complicating acute IWMI chest 1976 ; 69;599
20. Sujiura , T,Iwasaka ; T.Hasegaua, et al factors associated with persistent and transient fascicular blocks in anterior wall MI. AM J cardio 1989 ; 63 >84
21. Goldberg, R.prognosis of acute MI complicated by complete heart block the Worcester Heart attack study AM J cardio J 1992 ; 69; 1135
22. Hindman M. The clinical significance of bundle branch block complicating acute MI. clinical characteristics, hospital mortality and one year follow up , circulation 1978 ; 58;679
23. Hindman M. The clinical significance bundle branch block complicating acute MI..Indications of temperory and permanent pacemaker insertion.circulation 1978 ;58;679

24. Scheiman, MM, Gonzalez. R.P. fascicular block and acute MI JAMA 1980 ; 244;2646

25. Dubois , C, Pieland; LA Smeets , JP et al Short and long term prognostic importance of complete bundle branch block complication .acute MI Cli.card 1988 11;292

26. Lamas G. A simplified approach to predict the occurrence of CHB during acute MI AMJ Cardiolo. 1986;57;1213

27. Berger . PB. Ryan .TJ Inferior MI, High risk subgroups circulation 1990;81;401.

28. Nicad, P,Gilpin E. Dittrich , H et al Long term outcome in patients with inferior wall MI and complicate AV block J am coll Cardiol.1988;12;589

29. Behar S ZissmanE. Goldbourt , U et al Complicating Inferior wall myocardial infarction ; Short and long term prognosis. Am heart J 1993 ; 125; 1622

30. Ryan , TJ, Antman, EM , Brooks, NH et al 1998 update ;AC/C/AHA guidelines for the management of patients with acute myocardial infarction . A report of the American college of cardiology/ American heart association Task force on practice guidelines circulation 1999;100;1016

31. Ricou , MF ,Nicod,P,Gilpin et al . Influence of Right bundle branch block on short and long term survival after acute Myocardial infarction J Am coll cardiol1991;17;858

32. Milton et al arrythmias in acute MI published in chest 1964;45 pp 616-624

33. Nicod .P.et al factors associated with acute MI, J AM coll cardiol 1988;12;pp586

34. Dutta A.L.Das.S. Panda. M. Basu.J. profile of cardiac involvement in diabetes Journal of diabetes Association of India ;16;43-50.1976

35. Rodriguez-Morcu M et al :ECG Changes and cardiovascular risk factors in Type II diabetic patients.Salvd--- publica Mex—1999 Jan- Feb (1)12.7

36. Thraindottir is et al the epidemiology of right bundle branch block in association with cardiovascular mortality; Europian heart journal 1993 Dec 14(12) 1590-96

37. Panja .m. Dutta et al ; Study of 300 cases of chronic heart block – a clinical profile – Indian heart journal 33,83-900 1981

38. Partiman J.o.. Bradley .R.F. Acute myocardial infarction in 250 cases of diabetes , immediate mortality and five year survival ; New England Journal of Medicine 273; 455-461,1965

39. Nicod P et al factors associated with acute onset of AV block in acute MI. Jam coll cardiol. 1988;12;pp586

40. Goldberg et al prognosis of acute MI complicated by complete heart block; the Worcester heart attack . study AM J coll cardiol 1992 ; 69(14) pp1135-1141

41. Newby KH,Pisano , E Krucoff.MW et al Incidence and clinical relevance of the occurrence of bundle branch block in patients treated with thermolytic therapy. Circulaton 1996 ; 94;2424

CONSENT FORM

1. I agree to participate in the study titled " CONDUCTION DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION IN A SERIES OF 250CASES"

2. I confirm that I have been told about this study in my mother tongue and have had the opportunity to ask questions

3. I understand that my participation is voluntary and I may refuse to participate at any time without giving any reason and without affecting my benefits

4. I agree not to restrict the use of any data / results that arise from this study

Name of the participant

Signature /Thumb print

Witness: Investigator:

PROFORMA:

1. Name:

2. Age:

3. Sex:

4. Marietal status:

5. No of children:

6. Educational status:

7. Occupation:

8. Family income/yr:

9. Address:

10. Ht in cms:

11. Wt in kgs:

12. BMI [kg/m2].

13. Clinical presentation on admission:

14. Time of onset of symptoms:

Chest pain Typical/Atypical.

Syncope.

Palpitation.

Shortness of breath.

Cerebral symptoms.

PAST HISTORY:

Diabetes:

Hypertension:

Myocardial infarction:

Angina pectoris:

Cerebrovascular diseases:

PERSONAL HISTORY:

Diet: vegetarian/non vegetarian:

Smoking:

Alcohol:

Tobacco chewing:

Sedentary habits:

Menstrual and Obstetric History:

FAMILY HISTORY:

Hypertension:

Diabetes:

Ischemic heart disease:

Hypercholesteremia:

GENERAL EXAMINATION:

Presence of

Anemia:

Jaundice:

Cyanosis:

Clubbing:

Edema:

EXAMINATION OF CARDIOVASCULAR SYSTEM:

Pulse:

Rate.

Rhythm.

Character.

Volume.

Peripheral pulses.

BP:

JVP:

APICAL IMPULSE:

AUSCULTATION:

Heart sounds:

Murmur:

EXAMINATION OF RESPIRATORY SYSTEM:

Air entry:
Adventitious sounds:

EXAMINATION OF ABDOMEN:

Free fluid

Organomegaly

EXAMINATION OF CENTRAL NERVOUS SYSTEM:

Level of consciousness:

Any focal neurological deficit:

INVESTIGATIONS

1. Urine R/E.
   Albumin

   Sugar

   Deposits

2. BLOOD
   TC

   DC

   ESR

   HB

   Sugar

   Urea

3. ECG ANALYSIS.

   1. Site of infarction.

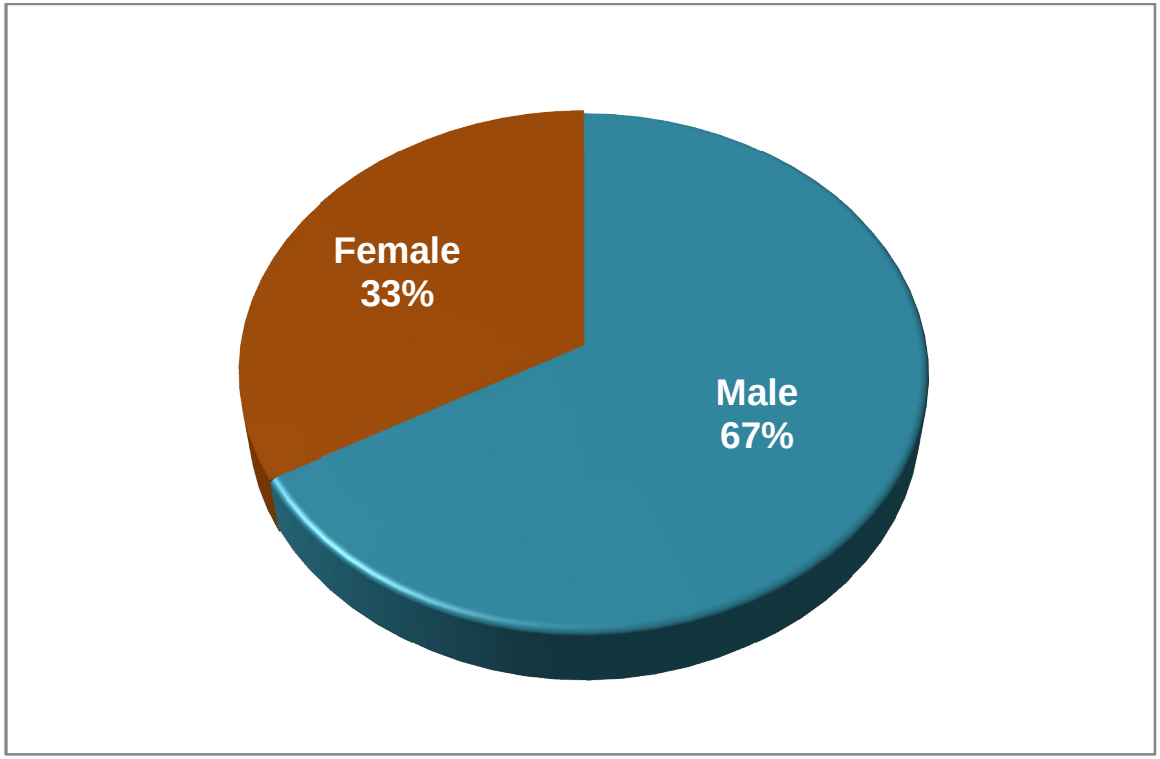
   2. Depth of infarction.

   3. Type of conduction disturbance present.

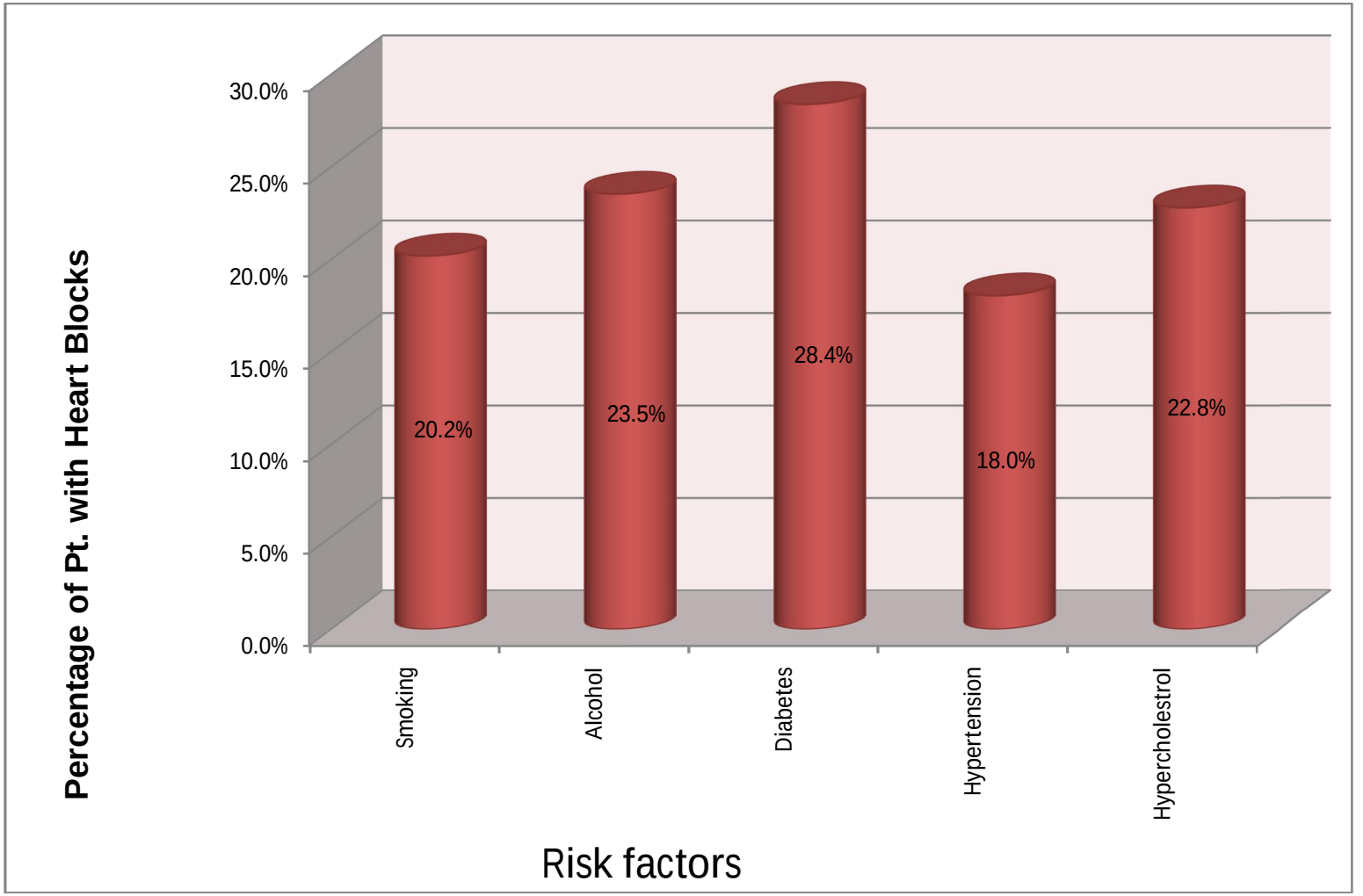
   4. Time of conduction disturbance with relation to onset of symptoms.

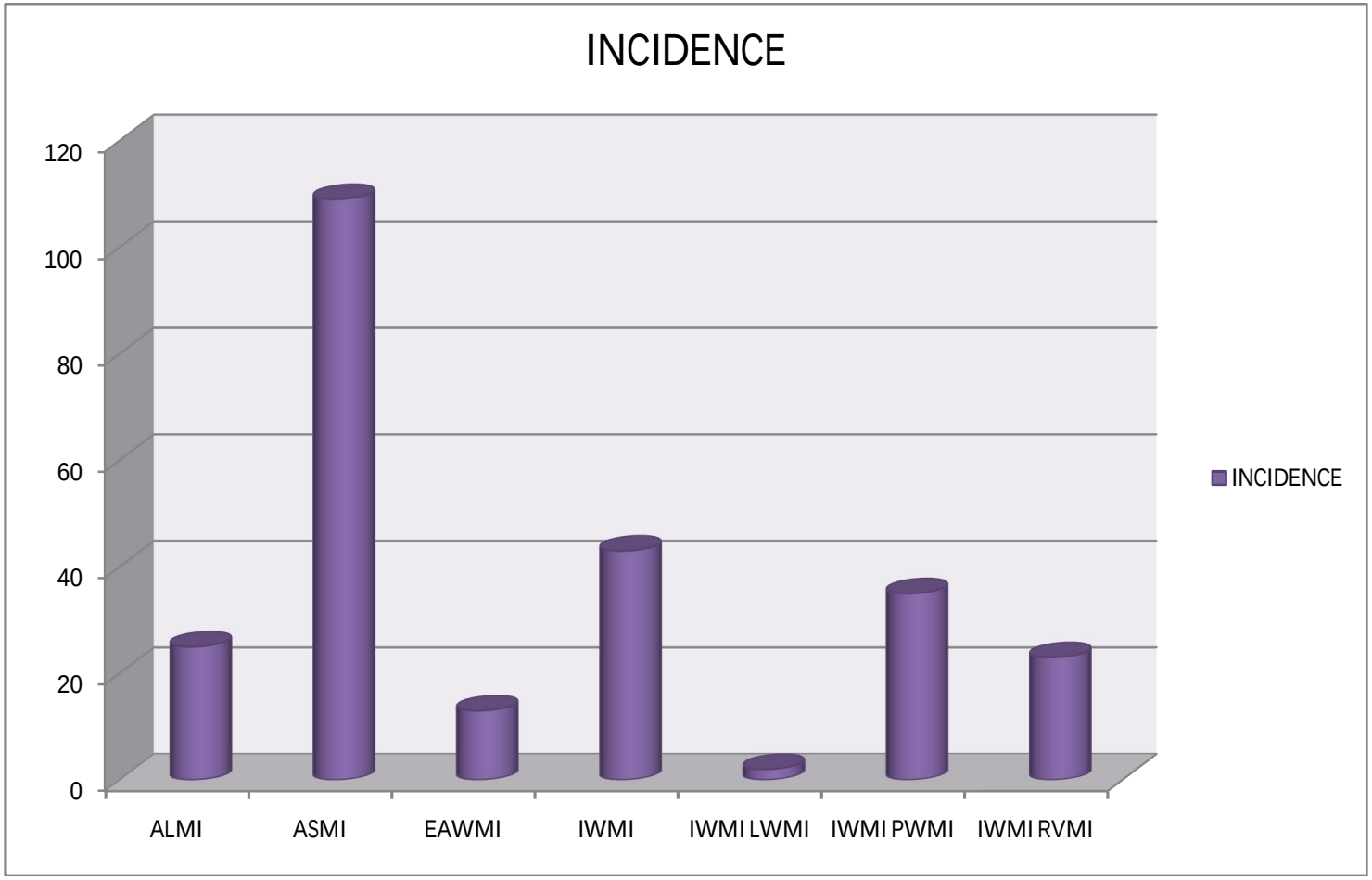
   5. Duration of conduction disturbance.
   6. Specific treatment given.

SEX DISTRIBUTION OF BLOCKS IN ACUTE MI

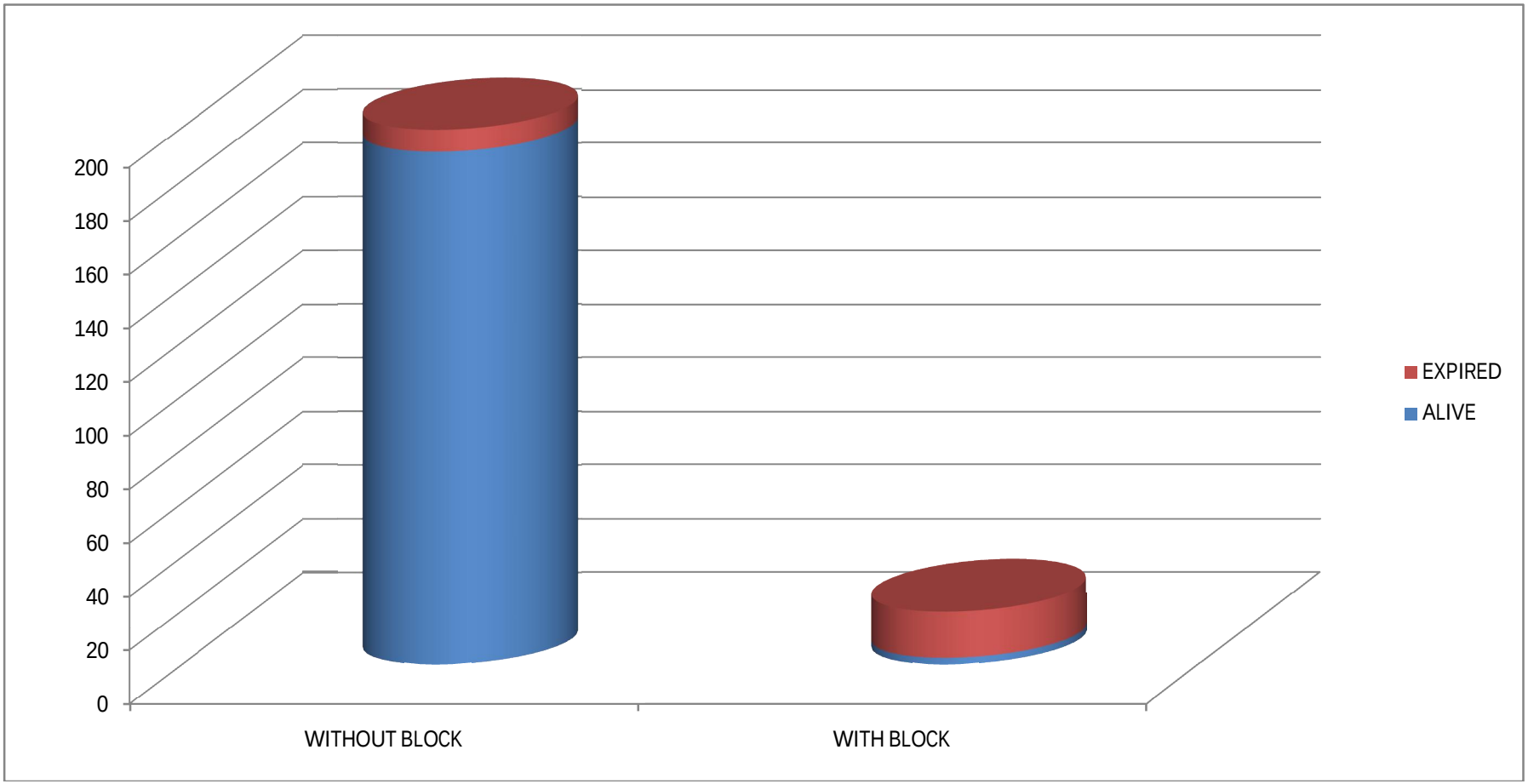


RISK FACTORS ASSOCIATED WITH HEART BLOCK

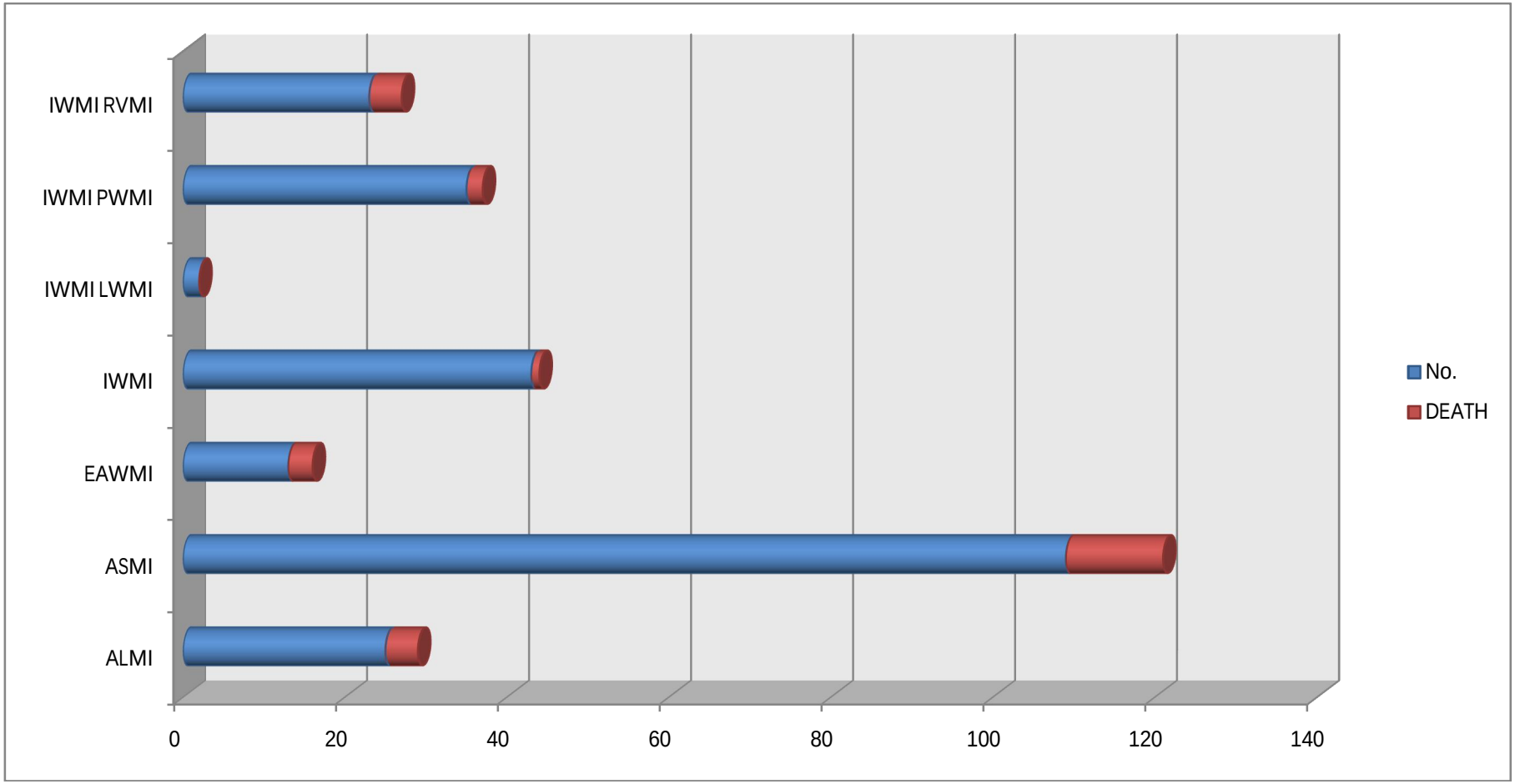



INCIDENCE
120
100
80
60
40
20
0
ALMI
ASMI
EAWMI
IWMI
IWMI LWMI
IWMI PWMI
IWMI RVMI
INCIDENCE

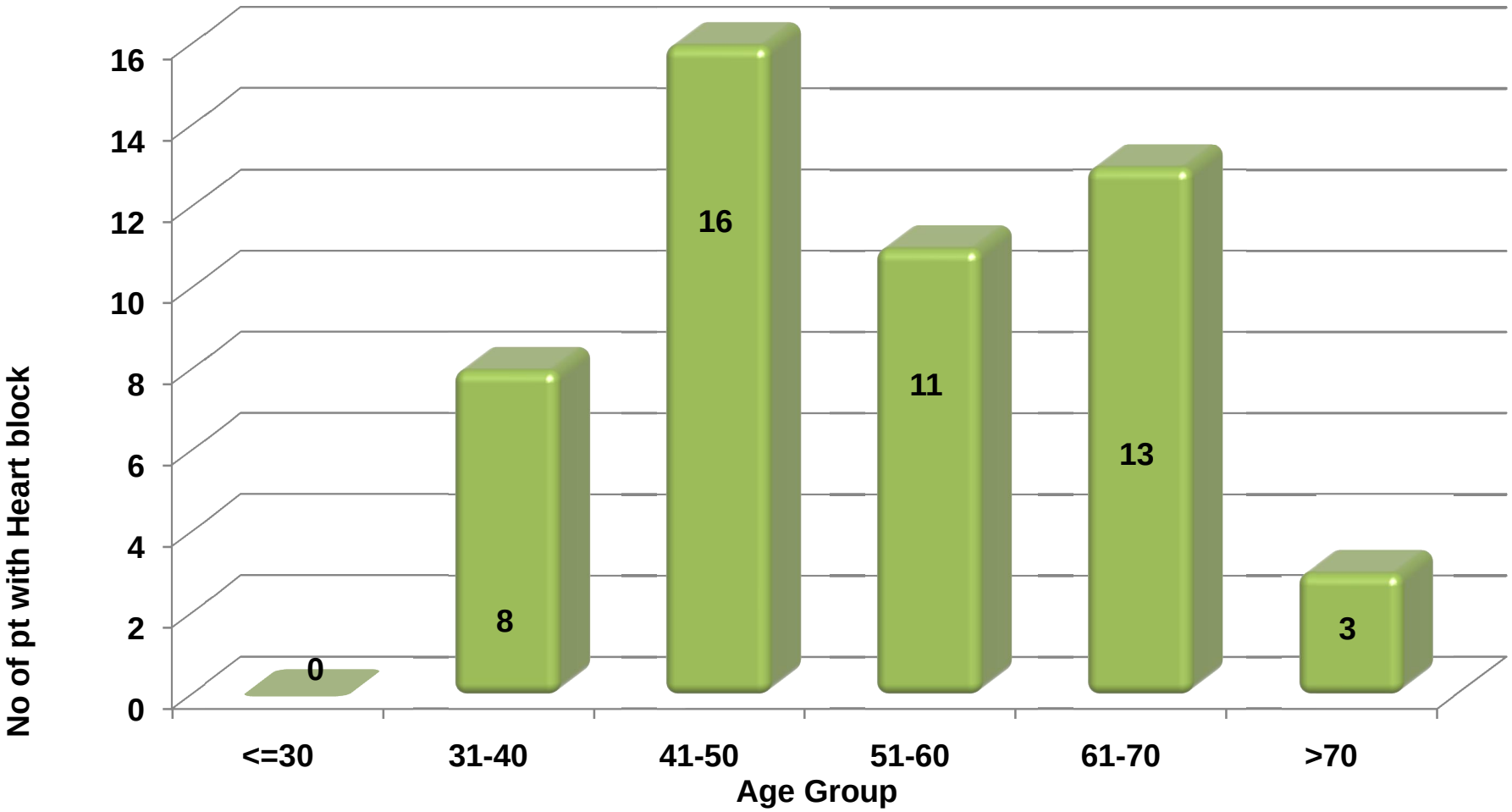
MORTALITY IN HEART BLOCKS



MORTALITY IN VARIOUS MI



AGE WISE DISTRIBUTION OF BLOCKS



Master Chart

S.NO	NAME	AGE	SEX	SMOKING	ALCOHOL	DIABETES	HYPERTENSION	HIGH CHO	TYPE OF MI	AV BLOCK	IVCD	THROMBOLISED	OUTCOME
1	Balakrishnan	39	M	YES	YES	NO	NO	YES	ASMI	0	0	YES	0
2	Dilli	60	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
3	Rajan	40	M	YES	YES	YES	YES	YES	ALMI	0	LAFB	YES	0
4	Alamelu	40	F	NO	NO	NO	YES	NO	ASMI	0	0	YES	0
5	Durai	50	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
6	Radha	65	F	NO	NO	NO	YES	NO	ASMI	0	0	YES	0
7	Kamala	55	F	NO	NO	YES	NO	YES	IWMI PWMI	0	0	YES	0
8	Indira bai	73	F	NO	NO	YES	YES	NO	ASMI	0	0	NO	0
9	Manirajan	63	M	NO	NO	NO	NO	NO	EAWMI	0	0	YES	0
10	Nagaraj	40	M	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
11	Jayakumar	41	M	YES	YES	NO	NO	YES	IWMI	0	0	YES	0
12	Ravi	35	M	NO	NO	YES	YES	YES	IWMI	0	0	YES	0
13	Gopal	65	M	NO	NO	NO	NO	NO	EAWMI	0	RBBB	NO	EXPIRED
14	Ravi	47	M	YES	YES	NO	NO	NO	ASMI	0	RBBB	YES	0
15	Amsa	40	F	NO	NO	YES	NO	YES	IWMI	0	0	YES	0
16	Jayakumar	39	M	YES	YES	NO	NO	NO	EAWMI	0	0	YES	0
17	Velayudam	70	M	YES	YES	NO	NO	YES	IWMI PWMI	0	0	NO	0
18	Nagaraj	48	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
19	Banni	50	M	NO	NO	NO	YES	NO	ASMI	0	0	YES	0
20	Abdul	65	M	YES	YES	NO	NO	NO	ASMI	0	0	NO	0
21	Sundar raj	44	M	NO	YES	NO	NO	NO	IWMI PWMI	0	0	YES	0
22	Vasanthi	53	F	NO	NO	NO	NO	NO	ASMI	0	LAFB	YES	EXPIRED
23	Chitrarasan	30	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
24	Dilly basha	60	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
25	Mohadeen	54	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
26	Rasathi	45	F	NO	NO	NO	YES	NO	ASMI	0	0	YES	0
27	Robert	52	M	YES	YES	NO	NO	NO	IWMI	0	0	YES	0
28	Elangovan	35	M	YES	YES	YES	YES	YES	EAWMI	0	0	YES	0
29	Kumar	39	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
30	Balasubramanium	62	M	NO	NO	YES	NO	NO	IWMI	0	LBBB	YES	0
31	Narayanan	60	M	NO	NO	YES	NO	NO	IWMI PWMI	0	0	YES	0
32	Muthu	39	M	NO	NO	YES	NO	NO	ASMI	0	0	YES	0
33	Kannappan	65	M	YES	YES	YES	YES	NO	ASMI	0	RBBB	NO	EXPIRED
34	Anbu	43	F	NO	NO	YES	YES	YES	ALMI	0	0	YES	0
35	Subramani	47	M	YES	YES	NO	NO	NO	IWMI	0	0	YES	0
36	Sasibegan	44	M	YES	YES	NO	NO	NO	IWMI PWMI	0	0	YES	0
37	Kumar	63	M	YES	YES	YES	NO	NO	EAWMI	0	0	YES	EXPIRED
38	Chandran	60	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
39	Nandakumar	54	M	NO	YES	YES	NO	NO	IWMI PWMI	0	RBBB	YES	0
40	Kurseeth begam	60	F	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
41	Shajagan	60	M	NO	YES	NO	YES	NO	IWMI PWMI	0	0	NO	0
42	Krishnan	43	M	NO	YES	NO	YES	NO	IWMI RVMI	0	RBBB	YES	EXPIRED
43	Baskaran	44	M	NO	NO	YES	NO	YES	IWMI LWMI	0	0	YES	0
44	Murali	44	M	YES	YES	YES	NO	YES	IWMI PWMI	0	0	YES	0
45	Ravikumar	50	M	YES	NO	NO	NO	NO	IWMI	0	RBBB	YES	0
46	Rajan	32	M	YES	YES	NO	NO	NO	ALMI	0	LBBB	YES	0
47	Laxmikantham	60	F	NO	NO	YES	YES	YES	ASMI	0	0	NO	0
48	Loganathan	47	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
49	Sajoja	65	F	NO	NO	YES	NO	NO	ASMI	0	LAFB	YES	0
50	Joseph	65	M	NO	NO	NO	NO	NO	ASMI	0	0	NO	0

Master Chart

S.NO	NAME	AGE	SEX	SMOKING	ALCOHOL	DIABETES	HYPERTENSION	HIGH CHO	TYPE OF MI	AV BLOCK	IVCD	THROMBOLISED	OUTCOME
51	Adhilaxmi	75	F	NO	NO	YES	YES	YES	IWMI	0	0	NO	0
52	Annabakyam	57	F	NO	NO	YES	YES	NO	IWMI	0	0	YES	0
53	Sujatha	60	F	NO	NO	YES	YES	NO	IWMI PWMI	0	0	YES	0
54	Shanthi	52	F	NO	NO	NO	NO	NO	ASMI	0	0	YES	EXPIRED
55	Angamma	70	F	NO	NO	NO	NO	YES	ASMI	0	0	NO	0
56	Krishnamoorthy	60	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
57	Elangovan	55	M	YES	YES	YES	YES	YES	IWMI PWMI	0	0	YES	0
58	Jaganathan	60	M	YES	YES	YES	NO	YES	IWMI PWMI	0	0	YES	0
59	Murugesan	65	M	YES	YES	NO	YES	NO	ASMI	0	0	YES	EXPIRED
60	Ranganathan	70	M	YES	NO	NO	NO	NO	IWMI RVMI	0	0	NO	0
61	Thangavel	54	M	YES	YES	NO	NO	NO	IWMI RVMI	0	0	YES	0
62	Nagalingam	60	M	NO	NO	YES	NO	YES	IWMI	0	0	YES	0
63	Meeramoideen	65	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
64	Fizarabi	50	M	NO	NO	YES	YES	YES	ASMI	0	LBBB	YES	EXPIRED
65	Mani	50	M	NO	NO	NO	NO	NO	IWMI RVMI	0	0	NO	0
66	Venkatesh	45	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
67	Mumtaj	68	F	NO	NO	YES	NO	YES	ALMI	0	0	YES	0
68	Danabakyam	54	F	NO	NO	NO	NO	NO	IWMI RVMI	0	0	YES	0
69	Jeyaraman	60	M	YES	NO	NO	NO	NO	IWMI PWMI	0	0	YES	0
70	Nagaiah	60	M	YES	NO	NO	YES	NO	IWMI	0	0	YES	0
71	Kannaiyah	65	M	NO	YES	NO	YES	NO	EAWMI	0	0	NO	0
72	Jinna	62	F	YES	YES	NO	NO	NO	IWMI PWMI	1 DEG	0	YES	0
73	Durasamy	45	M	YES	YES	NO	NO	NO	IWMI PWMI	0	RBBB	YES	EXPIRED
74	Ratnaraj	73	M	NO	NO	YES	YES	YES	ASMI	0	0	YES	0
75	Lenin raj	23	M	YES	NO	NO	NO	NO	IWMI RVMI	0	0	YES	0
76	Subramani	52	M	YES	YES	NO	NO	NO	ALMI	0	0	YES	0
77	Vijaya	36	F	NO	NO	YES	NO	YES	IWMI RVMI	0	0	YES	0
78	Sankar	35	M	NO	NO	NO	NO	YES	ASMI	0	LBBB	YES	0
79	Paramananda	55	M	YES	YES	YES	NO	YES	ASMI	0	LBBB	YES	EXPIRED
80	Kirubakaran	68	M	NO	NO	YES	YES	NO	IWMI RVMI	0	LAFB	YES	EXPIRED
81	Kandasami	62	M	NO	YES	YES	NO	YES	ASMI	0	0	YES	0
82	Laxmi	84	F	NO	NO	NO	NO	NO	EAWMI	0	0	YES	0
83	Baskar	60	M	YES	NO	NO	NO	NO	IWMI RVMI	0	0	YES	0
84	Danam	70	F	NO	NO	YES	NO	NO	IWMI RVMI	0	0	YES	0
85	Rubathy	45	F	NO	NO	NO	NO	NO	ALMI	0	0	YES	0
86	Aarumugam	53	M	YES	NO	NO	NO	NO	IWMI RVMI	1 DEG	0	YES	0
87	Thayaramal	60	F	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
88	Selvam	50	M	YES	YES	NO	NO	YES	IWMI PWMI	0	0	YES	0
89	Synambee	60	F	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
90	Gowseebee	60	F	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
91	Sivaganthi	60	M	YES	YES	NO	YES	NO	ASMI	0	0	YES	0
92	Kandasami	50	M	YES	YES	NO	NO	NO	ASMI	0	0	NO	0
93	Subash	45	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
94	Elumalai	50	M	NO	NO	YES	NO	NO	ASMI	0	0	YES	0
95	Aamirkhan	70	M	YES	YES	YES	NO	YES	ASMI	0	RBBB	YES	0
96	Kuppulaxmi	60	F	YES	NO	NO	YES	NO	IWMI	0	0	YES	0
97	Francis	50	M	YES	NO	NO	NO	NO	IWMI	0	0	YES	0
98	Anbazhagan	50	M	YES	YES	NO	NO	NO	EAWMI	0	0	YES	0
99	Baskar	34	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
100	Munusami	42	M	YES	YES	NO	NO	NO	IWMI PWMI	0	0	YES	0

Master Chart

S.NO	NAME	AGE	SEX	SMOKING	ALCOHOL	DIABETES	HYPERTENSION	HIGH CHO	TYPE OF MI	AV BLOCK	IVCD	THROMBOLISED	OUTCOME
101	Abdulsadar	70	M	NO	NO	NO	NO	YES	ASMI	0	0	NO	0
102	Asif	39	M	NO	NO	NO	NO	YES	IWMI RVMI	1 DEG	0	YES	0
103	Ramu	30	M	NO	NO	YES	NO	YES	IWMI PWMI	0	0	YES	0
104	Govindasamy	70	M	NO	NO	NO	NO	NO	ALMI	0	0	YES	0
105	Aziz	61	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
106	Krisnan	50	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
107	Agbarbasha	51	M	YES	YES	YES	NO	YES	ALMI	0	0	YES	0
108	Ravi	43	M	NO	NO	YES	NO	NO	ASMI	0	0	YES	0
109	Ibrahi	53	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
110	Laxmi	49	F	NO	NO	NO	NO	YES	ASMI	0	0	YES	0
111	Deivanai	75	F	YES	NO	NO	NO	NO	IWMI PWMI	0	0	YES	0
112	Egavalli	65	F	NO	NO	YES	YES	YES	ALMI	0	LBBB	YES	EXPIRED
113	Radha	53	M	NO	NO	NO	NO	NO	ALMI	0	0	YES	0
114	Krishnan	50	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
115	Basha	45	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
116	Vinodh	40	M	YES	YES	YES	NO	YES	ASMI	0	RBBB	YES	0
117	Alphones	50	M	YES	YES	NO	NO	NO	IWMI RVMI	0	0	YES	0
118	Perumathal	62	F	NO	NO	YES	NO	YES	IWMI RVMI	3 DEG	0	YES	EXPIRED
119	Muthumani	40	F	NO	NO	NO	NO	NO	IWMI PWMI	3 DEG	0	YES	0
120	Logammal	62	F	NO	NO	NO	NO	NO	ALMI	0	RBBB	YES	EXPIRED
121	Kuppammal	70	F	NO	NO	YES	YES	YES	ASMI	0	0	NO	0
122	Suseela	45	F	NO	NO	YES	NO	NO	IWMI	0	0	YES	0
123	Gurusamy	54	M	YES	YES	YES	NO	NO	IWMI PWMI	0	0	YES	0
124	Vinodh	40	M	YES	NO	YES	NO	YES	ASMI	0	RBBB	YES	0
125	Revathy	49	F	NO	NO	YES	NO	NO	ASMI	0	0	YES	0
126	Subramni	71	M	NO	NO	YES	YES	YES	ALMI	0	0	NO	0
127	Radaiyah	64	M	NO	NO	NO	NO	NO	IWMI PWMI	0	0	YES	0
128	Sumathi	36	F	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
129	Selvaraj	50	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
130	Saroja	35	F	NO	NO	YES	YES	YES	IWMI	0	LBBB	YES	EXPIRED
131	Venugopal	45	M	NO	NO	NO	NO	NO	ASMI	3 DEG	0	YES	EXPIRED
132	Deivanai	75	F	NO	NO	YES	YES	YES	IWMI PWMI	3 DEG	0	NO	0
133	Madhavan	43	M	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
134	Periyasami	52	M	YES	NO	YES	NO	NO	IWMI	0	0	YES	0
135	Sriram	42	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
136	Sathya	63	M	NO	NO	YES	YES	YES	ASMI	0	0	YES	0
137	Mahalaxmi	49	F	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
138	Danam	70	F	NO	NO	NO	YES	YES	IWMI PWMI	0	0	NO	0
139	Gothanarama	48	M	YES	NO	NO	YES	NO	ALMI	0	0	YES	0
140	Laxmi	84	F	YES	NO	YES	NO	NO	ASMI	0	0	YES	0
141	Raman	70	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
142	Venkatesh	45	M	YES	YES	NO	YES	NO	IWMI	0	0	YES	0
143	Madavan	55	M	YES	YES	NO	YES	YES	ASMI	0	0	YES	0
144	Selvam	62	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
145	Kumar	72	M	NO	NO	NO	YES	YES	IWMI	2 DEG	0	YES	0
146	Selvi	42	F	NO	NO	YES	NO	NO	ASMI	0	0	YES	0
147	Thayammal	52	F	NO	NO	NO	NO	YES	ASMI	0	0	YES	0
148	Mari	62	F	NO	NO	NO	YES	NO	IWMI	0	0	NO	0
149	Kokila	72	F	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
150	Laxmi	39	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0

Master Chart

S.NO	NAME	AGE	SEX	SMOKING	ALCOHOL	DIABETES	HYPERTENSION	HIGH CHO	TYPE OF MI	AV BLOCK	IVCD	THROMBOLISED	OUTCOME
151	Krishnan	50	M	NO	NO	YES	YES	YES	ASMI	0	LBBB	YES	0
152	Rajammal	70	F	NO	NO	NO	NO	YES	ASMI	0	0	YES	EXPIRED
153	Alagesan	70	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
154	Ravi	65	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
155	Sarathmary	60	F	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
156	Mary	75	F	NO	NO	YES	YES	YES	IWMI PWMI	3 DEG	0	NO	0
157	Palaniappan	60	M	YES	YES	NO	YES	NO	ASMI	0	0	YES	EXPIRED
158	Laxmi	62	F	YES	YES	NO	NO	NO	IWMI PWMI	1 DEG	0	YES	0
159	karpagam	53	F	NO	NO	NO	NO	NO	ASMI	0	LAFB	YES	0
160	Santhanam	52	M	YES	YES	NO	NO	NO	IWMI	0	0	YES	0
161	Radakrishnan	50	M	YES	YES	NO	NO	YES	IWMI PWMI	0	0	YES	0
162	Nagammal	45	F	NO	NO	NO	NO	NO	ALMI	0	0	YES	0
163	Sakirunisha	40	F	NO	NO	NO	YES	NO	ASMI	0	0	YES	0
164	Pandian	54	M	NO	YES	YES	NO	NO	IWMI	0	RBBB	NO	0
165	Israel	53	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
166	Sornamal	62	F	NO	NO	NO	YES	NO	IWMI	0	0	YES	0
167	Balaji	71	M	NO	NO	YES	YES	YES	ALMI	0	0	NO	0
168	Muthu	60	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
169	Venkatesh	60	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
170	Valliammal	49	F	NO	NO	YES	NO	NO	ASMI	0	0	YES	0
171	Jeyaraman	51	M	YES	YES	YES	NO	YES	ALMI	0	BB	YES	0
172	Subramani	50	M	YES	YES	NO	NO	NO	IWMI RVMI	0	0	YES	0
173	Vijaylaxman	65	M	NO	YES	NO	NO	NO	ASMI	0	0	YES	0
174	Jai	55	M	YES	YES	YES	YES	YES	IWMI PWMI	0	0	YES	0
175	Krishnaveni	84	F	NO	NO	NO	NO	NO	EAWMI	0	0	NO	0
176	Subramani	45	M	YES	YES	NO	NO	NO	ASMI	0	RBBB	YES	0
177	Abdullah	47	M	YES	YES	NO	NO	NO	ASMI	0	RBBB	YES	EXPIRED
178	Seshammal	45	F	NO	NO	NO	NO	NO	ALMI	0	0	YES	0
179	Nagaraj	44	M	YES	YES	NO	NO	NO	IWMI PWMI	0	0	YES	0
180	Samundeeswari	40	F	NO	NO	YES	NO	YES	IWMI	0	0	YES	0
181	Jeya	39	F	NO	NO	NO	NO	YES	IWMI RVMI	0	0	YES	0
182	Venkatesan	63	M	NO	NO	NO	NO	NO	EAWMI	0	0	YES	EXPIRED
183	Pitchaiyah	40	M	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
184	Devi	60	F	NO	NO	NO	NO	NO	ASMI	0	LAFB	NO	0
185	Selvaraj	50	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
186	Ganapathy	62	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
187	Sanmughadurai	44	M	NO	YES	NO	NO	YES	IWMI LWMI	0	0	YES	0
188	Meena	49	F	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
189	Selvam	47	M	YES	YES	NO	NO	NO	ALMI	0	BB	YES	EXPIRED
190	Lalitha	75	F	NO	NO	YES	YES	YES	IWMI	0	0	NO	0
191	Aathikesavalu	60	M	NO	NO	YES	NO	YES	IWMI	0	0	YES	0
192	Nagarajan	65	M	NO	YES	NO	YES	YES	ALMI	0	0	YES	0
193	Arumugam	60	M	NO	NO	NO	NO	YES	IWMI PWMI	0	0	YES	0
194	Peterson	65	M	NO	NO	NO	NO	NO	ALMI	0	RBBB	YES	EXPIRED
195	Ramamurthy	45	M	YES	YES	NO	NO	NO	ASMI	0	LBBB	YES	0
196	Ameer	62	M	NO	YES	YES	NO	YES	ASMI	0	0	YES	0
197	Mariammal	68	F	NO	NO	YES	NO	YES	ALMI	0	0	YES	0
198	Pokkisham	36	F	NO	NO	NO	NO	NO	IWMI	0	0	YES	0
199	Danalaxmi	45	F	NO	NO	NO	YES	NO	ASMI	0	0	YES	0
200	Danalaxmi	60	F	NO	NO	NO	NO	NO	IWMI	0	RBBB	YES	0

Master Chart

201	Abdulrahman	70	M	NO	NO	NO	NO	NO	ALMI	0	0	NO	0
S.NO	**NAME**	**AGE**	**SEX**	**SMOKING**	**ALCOHOL**	**DIABETES**	**HYPERTENSION**	**HIGH CHO**	**TYPE OF MI**	**AV BLOCK**	**IVCD**	**THROMBOLISED**	**OUTCOME**
202	Kamala	73	F	NO	NO	YES	YES	NO	ASMI	0	0	YES	0
203	Ramani	52	F	NO	NO	NO	NO	YES	ASMI	0	0	YES	0
204	Rajan	62	M	NO	NO	NO	NO	NO	ALMI	0	0	YES	0
205	Revathy	43	F	NO	NO	NO	NO	NO	IWMI	0	LBBB	YES	0
206	raji	35	F	NO	NO	NO	NO	YES	ASMI	0	0	YES	0
207	Rajathi	70	F	NO	NO	YES	NO	NO	IWMI RVMI	0	0	YES	0
208	Shakeer ahmed	54	M	YES	NO	NO	NO	NO	IWMI PWMI	1DEG	0	YES	0
209	Kuppusami	54	M	YES	YES	NO	NO	NO	IWMI RVMI	0	0	YES	0
210	Anadhi	50	F	NO	NO	NO	NO	NO	ASMI	0	LAFB	YES	0
211	Syed	34	M	YES	YES	NO	NO	NO	ASMI	0	0	YES	0
212	Ramu	44	M	YES	YES	YES	NO	YES	IWMI PWMI	0	0	YES	0
213	Janaki	48	F	NO	NO	NO	NO	NO	ASMI	0	LBBB	YES	0
214	Arumugam	50	M	YES	NO	NO	NO	NO	IWMI	0	0	YES	0
215	Raju	42	M	YES	YES	NO	NO	NO	ASMI	0	LBBB	YES	0
216	Raman	50	M	YES	NO	NO	NO	NO	IWMI RVMI	0	RBBB	YES	EXPIRED
217	Muthu	65	F	NO	NO	NO	YES	NO	ASMI	0	0	YES	EXPIRED
218	Ravi	65	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
219	Xavier	40	M	YES	YES	YES	YES	YES	ALMI	0	0	YES	0
220	Devanathan	60	M	YES	NO	NO	YES	NO	IWMI	0	0	YES	0
221	Sampoornam	70	F	NO	NO	NO	YES	YES	IWMI PWMI	0	0	NO	0
222	Ramaiyah	39	M	YES	YES	NO	NO	YES	ASMI	0	0	YES	0
223	Vijayan	55	M	YES	YES	YES	NO	YES	ASMI	0	LBBB	YES	0
224	Jamayutheen	55	M	YES	NO	YES	YES	YES	ASMI	0	0	YES	0
225	Dandapani	45	M	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
226	Sanmugavel	70	M	NO	NO	NO	NO	YES	ASMI	0	0	YES	EXPIRED
227	Murthy	65	M	YES	YES	NO	YES	NO	ASMI	0	0	YES	0
228	Elumalai	50	M	NO	NO	NO	NO	NO	IWMI RVMI	0	0	YES	0
229	Savithri	54	F	NO	NO	NO	NO	NO	IWMI RVMI	0	0	YES	0
230	Kanagambari	72	F	NO	NO	NO	NO	NO	ASMI	0	0	YES	0
231	Vijaylakshmi	70	F	NO	NO	YES	YES	YES	ASMI	0	0	YES	0
232	Periasamy	50	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
233	Velammal	62	F	NO	NO	YES	NO	YES	IWMI RVMI	3 DEG	0	YES	0
234	Sekar	60	M	NO	YES	NO	YES	NO	IWMI PWMI	0	0	YES	0
235	Nayaki	35	F	NO	NO	YES	YES	YES	IWMI	0	0	YES	0
236	Dandapani	60	M	YES	NO	NO	NO	NO	IWMI RVMI	0	0	YES	0
237	Nalini	60	F	YES	NO	NO	YES	NO	IWMI	0	0	YES	0
238	Tamilselvan	35	M	YES	YES	YES	YES	YES	EAWMI	0	0	YES	0
239	Ilamaran	32	M	YES	YES	NO	NO	NO	ALMI	0	0	YES	0
240	Kamalakannan	52	M	YES	NO	NO	NO	YES	IWMI	0	0	YES	0
241	Lakshmi	55	F	NO	NO	YES	NO	YES	IWMI PWMI	0	0	YES	0
242	Raja	42	M	YES	YES	NO	NO	NO	IWMI PWMI	0	0	YES	0
243	Devan	47	M	YES	NO	NO	NO	NO	ASMI	0	0	YES	0
244	Kumar	45	M	YES	YES	NO	YES	NO	IWMI	0	0	YES	0
245	Alamelu	45	F	NO	NO	NO	NO	YES	IWMI	0	0	YES	0
246	Maran	65	M	NO	YES	NO	YES	NO	EAWMI	0	0	NO	0
247	Natesan	63	M	NO	NO	NO	NO	NO	EAWMI	0	0	YES	0
248	Sengotuvel	43	M	NO	NO	NO	NO	YES	ASMI	0	0	YES	0
249	Mani	41	M	YES	YES	NO	NO	YES	IWMI	0	0	YES	0
250	kumar	47	M	YES	YES	NO	NO	YES	ASMI	0	0	YES	0

CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

Dr.P.Arul, PG in MD(GM)

Dear Dr.P.Arul, PG in MD(GM)

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

"Conduction Disturbances in Acute myocardial infarction – A study of 250 cases on infarction "

The following members of the ethics committee were present at the meeting held on 28.01.2010 at the Council Hall, Stanley Medical College, Chennai-1 at 10.00AM

Dr.C.B.Tharani, Director of Pharmacology,
Madras Medical College, Chennai-3 - Chairman of the Ethics Committee
Dr.S. Chitra, Vice-Principal,
Stanley Medical College, Chennai – 1- Member Secretary of the Ethics Committee

MEMBERS

Dr.Jayanthi
Prof.of Medical Gastroenterology
Dr.Madhavan
Prof.of Pharmacology
Dr.E.Dhandapani
Prof.of Medicine
Dr.Sujatha Sridharan
Prof.of Paediatrics
Thiru.Pachaiappan,
Junior Administrative Officer,
Thiru.A. Senthil Manoharan,
Advocate

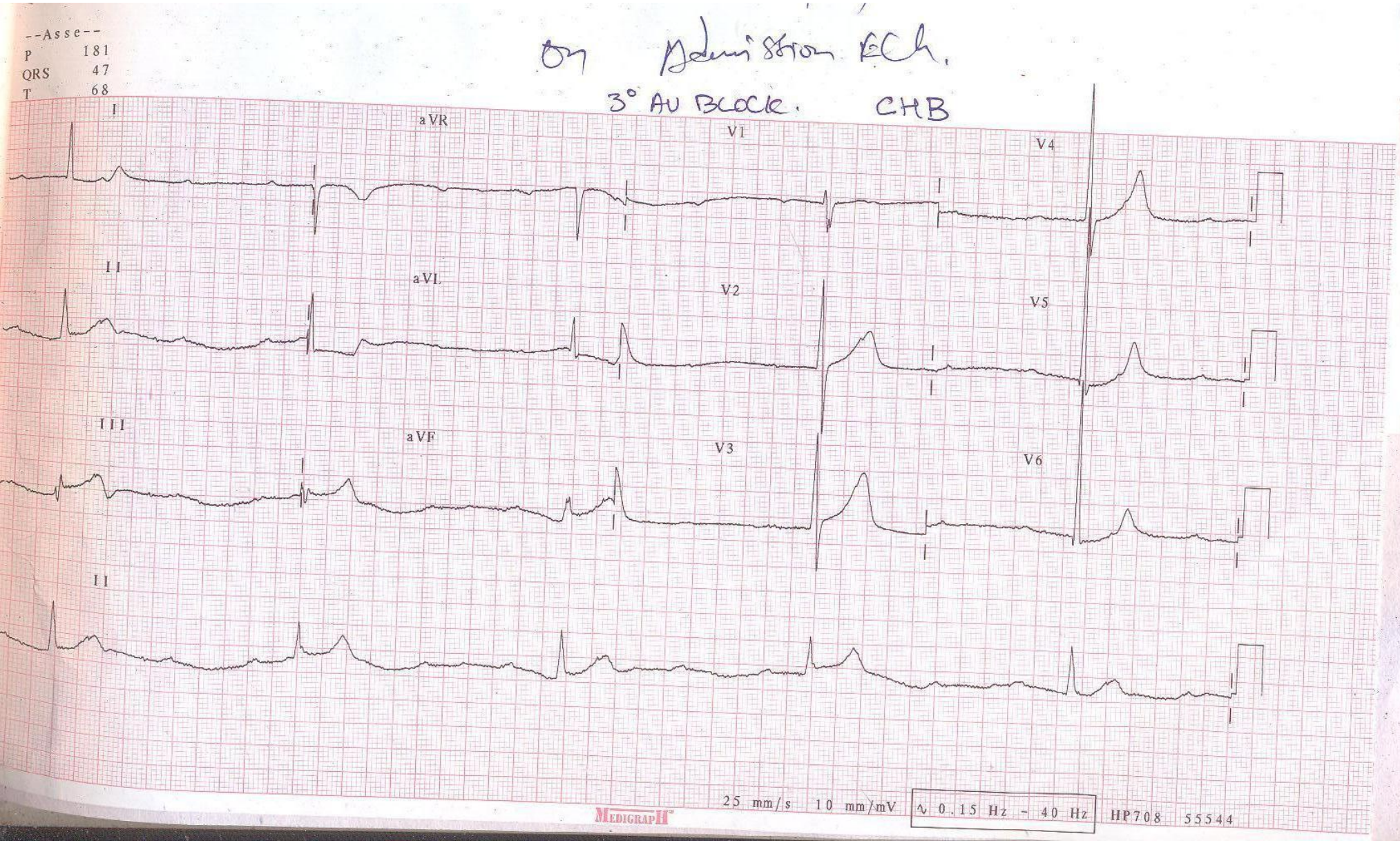
We approve the project to be conducted in its presented form.

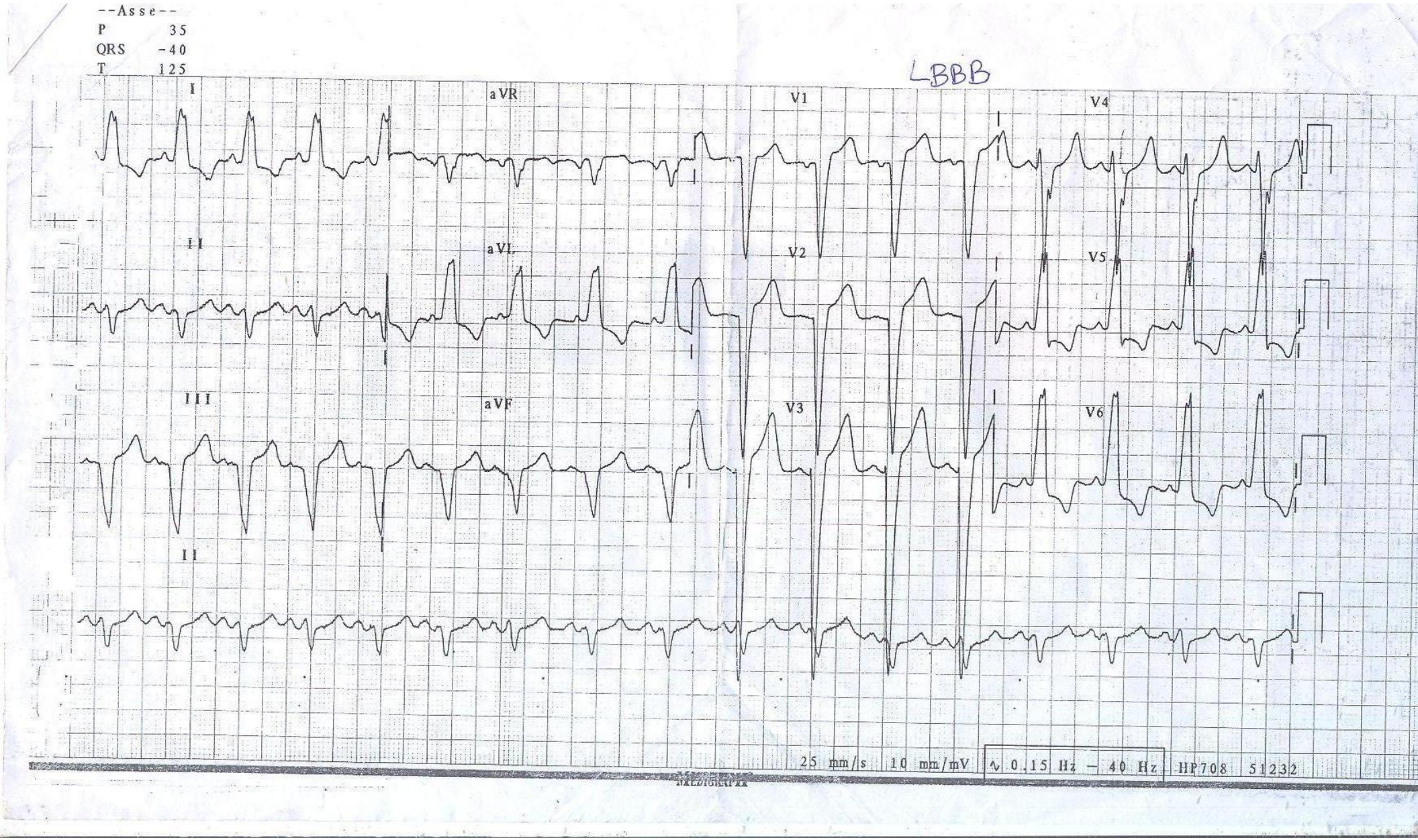
The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

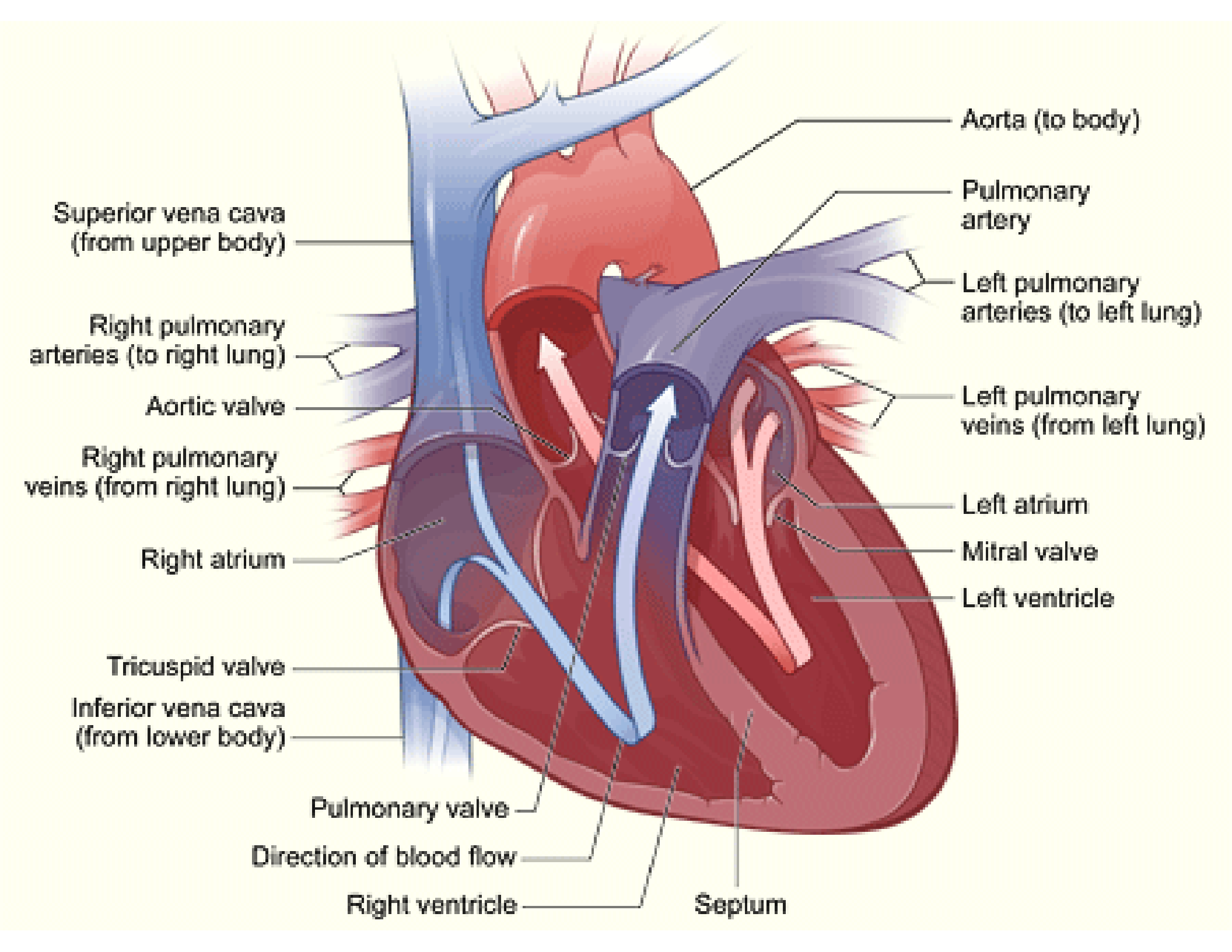
Yours sincerely,

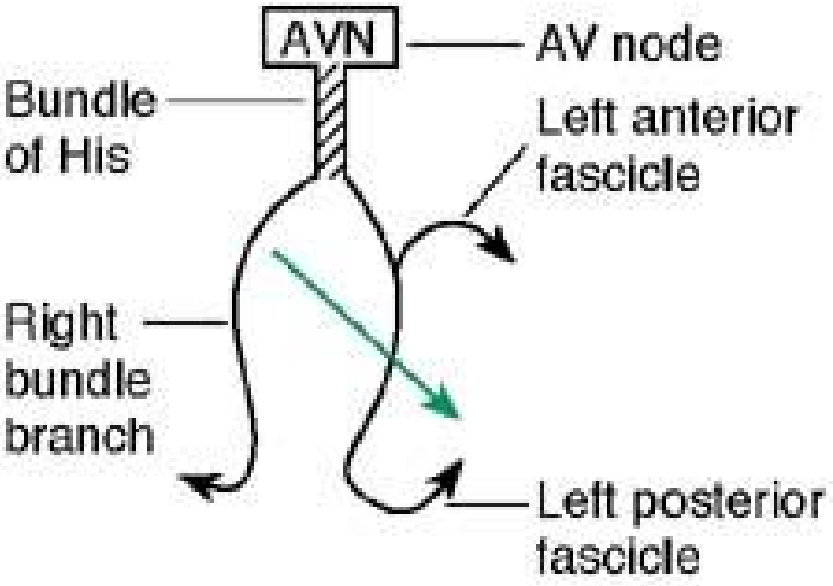
Member Secretary,
Ethics Committee

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

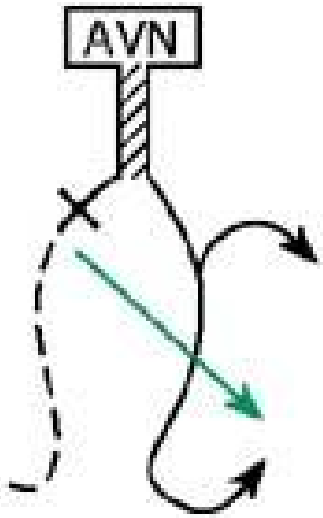

--Asse--
P 181
QRS 47
T 68
On Admission ECh.
3° AV BLOCK. CHB
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s 10 mm/mV
0.15 Hz - 40 Hz
HP708 55544
MEDIGRAPH


--Asse--
P 35
QRS -40
T 125
LBBB
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
25 mm/s 10 mm/mV ∿ 0.15 Hz – 40 Hz HP708 51232

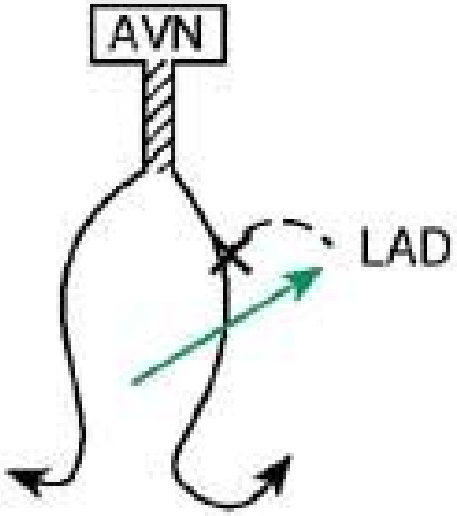

Aorta (to body)
Pulmonary artery
Superior vena cava (from upper body)
Left pulmonary arteries (to left lung)
Right pulmonary arteries (to right lung)
Left pulmonary veins (from left lung)
Aortic valve
Right pulmonary veins (from right lung)
Left atrium
Right atrium
Mitral valve
Left ventricle
Tricuspid valve
Inferior vena cava (from lower body)
Pulmonary valve
Direction of blood flow
Right ventricle
Septum



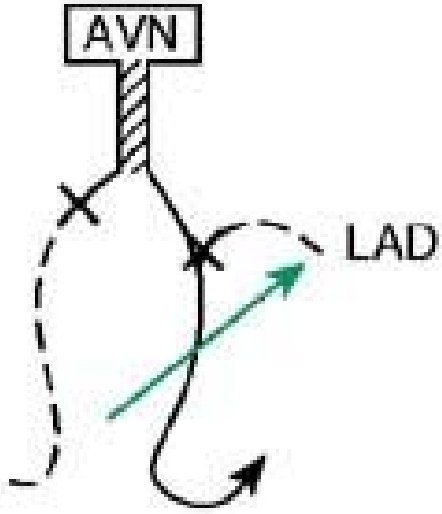
1. Normal conduction → normal axis



2. RBBB → normal axis (as left ventricle depolarizes normally)



3. Left anterior hemiblock → left axis deviation



4. RBBB and left anterior hemiblock → left axis deviation

After J Hampton *ECG Made Easy* Churchill Livingstone.